Report of the Royal Commission of Inquiry into Certain Deaths at the Hospital for Sick Children and Related Matters

The Honourable Mr. Justice
Samuel G. M. Grange
Supreme Court of Ontario
Commissioner

Ontario

Published by the Ontario Ministry
of the Attorney General

Printed in Canada - 1984

ISBN 0-7743-9968-6

Copies are available in person from:

Ontario Government Bookstore,
880 Bay Street
Toronto, Ontario

Or by mail through:

Publication Services
5th Floor
880 Bay Street
Toronto, Ontario
M7A 1N8

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December 28, 1984

The Honourable R. Roy McMurtry, Q.C.
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Dear Mr. Attorney:

Pursuant to the Orders-in-Council of April 21, 1983 and May 24, 1984, setting up this Royal Commission under the Public Inquiries Act, I have completed my Report and now submit it.

Yours very truly,

S. G. M. Grange
Commissioner

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TEPPERMAN, Dr. Paul	Coroner for the Province of Ontario
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A. INTRODUCTION

1. THE STORY IN OUTLINE

On June 30, 1980, Laura Woodcock, an eighteen day old baby girl, died on Ward 4B at the Hospital for Sick Children at Toronto. I shall sometimes hereafter refer in this Report to that hospital as the "Hospital".

Ward 4B was one of two cardiac wards of the Hospital, the other being 4A, which was adjacent to 4B, was associated with it, and had certain common facilities. The two wards are sometimes hereafter referred to as "Wards 4A/B". After the death of Laura Woodcock, thirty-two babies and three older children died on one or other of the wards during the months of July, 1980 to March, 1981, culminating in the death of fourteen week old Justin Cook on Ward 4A in the early morning of March 22, 1981. A list of all of these children is found on page 10, and I shall deal with them, their clinical conditions, and the circumstances of their deaths in detail later, but it is sufficient for the moment to say that by March 22, 1981, suspicions had been aroused that some of the babies had died from overdoses of digoxin, a drug used therapeutically for the control of congestive heart failure. On Sunday March 22, one of the teams of nurses regularly employed on Ward 4A was relieved of duty. By the following Friday one Susan Nelles, a member of that team of nurses, had been arrested and charged with the murder of Janice Estrella who died on Ward 4A on January 11, of Kevin Pacsai who died shortly after transfer from Ward 4B on March 12, of Allana Miller who died on Ward 4A on March 21, and of Justin Cook.

The Preliminary Inquiry into the charges against Susan Nelles commenced before His Honour Judge Vanek on January 11, 1982, and terminated after forty-five days of hearing with the discharge of the accused. The investigation into the deaths continued but no further arrests were made. On April 21, 1983, the Lieutenant Governor-in-Council appointed this Commission.

2. THE COMMISSION

My Terms of Reference, as will be seen, required me:

> . . . without expressing any conclusion of law regarding civil or criminal responsibility . . .
>
> 3) to inquire into and report on and make any recommendations with respect to how and by what means children who died in Cardiac Wards 4A and 4B at the Hospital for Sick Children between July 1st, 1980 and March 31st, 1981, came to their deaths;
>
> 4) to inquire into, determine and report on the circumstances surrounding the investigation, institution, and prosecution of charges arising out of the deaths of the above mentioned four infants.

At the outset we divided the hearings into two phases, Phase I being the matter referred to in paragraph 3) and Phase II being that in paragraph 4). I should first point out that while the direction in paragraph 3) (Phase I) covered the period only from July 1, 1980, and concerned only children who died on the wards, the death of Laura Woodcock who died on June 30, 1980, and that of Kevin Pacsai who died in the Intensive Care Unit (I.C.U.) were included in our investigation without objection from any party. Laura Woodcock's death was only one day outside the period and was strikingly similar to many of the others; Kevin Pacsai's onset of terminal events took place on Ward 4B. He was transferred to the I.C.U. where he died only six hours later. He was also one of the four babies whose deaths were the subject of the charges against Susan Nelles.

It is clear that the reference in paragraph 4) (Phase II) to "the above mentioned four infants" refers to the babies Estrella, Pacsai, Miller, and Cook. Less clear was when the period of investigation into which I was to inquire was to end. The investigation, which began on March 22, 1981, certainly continued until the early months of 1983 and, indeed, has still not been formally closed.

I ruled that we would stop at the date of the discharge of Susan Nelles, namely May 21, 1982, and that ruling was not challenged.

A much more difficult problem arose in the interpretation of paragraph 3) and that problem was whether I was entitled, if I should find that any person or persons had administered an overdose of digoxin to any child, to name that person or persons. I ruled that I was, subject to certain limitations, and that ruling was upheld in the Divisional Court. One judge - Reid J. - would have imposed a further limitation against stating whether the death was accidentally or deliberately caused. An appeal was taken to the Court of Appeal and that Court, by Judgment released April 12, 1984 (now reported 46 O.R. (2d) 210), reversed the Divisional Court and held that I was not entitled to attach a name to the administrator, whether the administration was accidental or designed, because if I were to do so, I would violate the prohibition in my Terms of Reference against the expression of a conclusion of law regarding civil or criminal responsibility.

The Judgment posed a problem for me. As it will be seen, there was toxicologic evidence suggesting an overdose of digoxin for only a very few of the children. One of the chief arguments put forward by those advocating findings of foul play in the deaths of other babies was the constant presence at the scene of the deaths of one or more members of a particular team of nurses. Any reference to that argument and any finding acceding to it might well be deemed at least a partial identification of the killer or killers and thus a breach of the injunction in my Terms of Reference as interpreted by the Court of Appeal. I reached the conclusion that I must consider that question regardless. The Reasons for Judgment state (infra, Appendix 3, page 18):

> The Commissioner is obliged to hear all of the evidence relating to the cause of the death of the children and this would include evidence which tended to show that one or more of them died as a result of unlawful or negligent acts. While the Commissioner must not identify an individual as being legally responsible for a death, he should analyze and report upon all of the evidence with respect to the circumstances of each death. . . .

Obviously one of the circumstances of the deaths is the constant presence of one or more members of that team. I must obey the one direction and not disobey the other. I must analyze the evidence relating to the circumstances of each death, which includes the presence of the nursing team, and if, taking that evidence into consideration, I should find that a death was caused by an overdose of digoxin, I must not attach a name to the administrator. The resulting balancing act is, in the words of the Court of Appeal (infra, Appendix 3, page 19), " . . . one of extreme difficulty at times approaching the impossible." I shall do my best bearing in mind the further direction of the Court (on the same page) that "Where such an impasse arises it should be resolved, in our opinion, by a course that best protects the civil rights of the persons the limitation was designed to protect." Perhaps there will be some evidence which has influenced me in reaching my conclusions upon which I can neither report nor comment.

A potentially more far-reaching difficulty arose in relation to the proceedings in Phase II. Susan Nelles had brought action against the Crown, the Attorney General, the Chief of Police of the Metropolitan Toronto Police Force, and certain officers of that Force claiming damages:

(a) for negligence in the investigation, in the laying of charges and in the prosecution;

(b) for breach of the Canadian Charter of Rights and Freedoms by allegedly relying upon the silence of Susan Nelles as evidence of guilt;

(c) for false imprisonment; and

(d) for malicious prosecution.

It may be that as a matter of law no action for negligence lies against the Police or the Crown in the circumstances of this case. Further, Miss Nelles' action, insofar as it asserts claims against the Crown and the Attorney General, has been struck out (unreported decision of Mr. Justice Fitzpatrick, August 19, 1983. At the time of writing, an appeal has been heard and is under reserve). Nevertheless, those causes of action have been asserted in Miss Nelles' action and I therefore have to be concerned with the statement found infra in the Reasons for Judgment of the Court of Appeal (Appendix 3, pages 14-15):

> But the Order-in-Council specifically limits the Commissioner by forbidding him to express any conclusion of law regarding civil or criminal responsibility . . . In our opinion, such a conclusion may be expressed by findings of fact which without more, when found against a named person, constitute a conclusion of criminal or civil responsibility.

My fear was that the Judgment might preclude my proceeding with Phase II at all. The Court of Appeal was, in the passage quoted, of course dealing with Phase I of the Inquiry, but the limitation applied to both Phases, and it was at least arguable that any finding by me of proper or improper action on the part of the Police, the Coroners, or the Crown Attorneys might, without more, be deemed a finding of civil responsibility or of the absence of it. Moreover, if I could not make such findings, I should not hear evidence which was relevant only to those issues. That essentially would leave nothing to be heard.

Indeed it was so argued. Mr. Percival for the Police stated categorically (as he had every right to do) that when the first question was asked with a view to showing any impropriety on the part of the Police he would object and, if I should rule against him, challenge that ruling in the Courts. Phase II was about to start. I did not view with favour the delay involved in such a course. I accordingly sought clarification from the Attorney General who caused the Order-in-Council to be amended on May 24, 1984. As amended, paragraph 4) of the Terms of Reference now reads:

> 4) to inquire into, determine and report on the circumstances surrounding the investigation, institution, and prosecution of charges arising out of the deaths of the above-mentioned four infants; and, without restricting the generality of the foregoing, the Commissioner may receive evidence and submissions and comment fully on the conduct

> of any person during the course of the investigation, institution, and prosecution of charges arising out of the deaths of the above-mentioned four infants, provided that such comment does not express any conclusion of law regarding civil or criminal responsibility. (Amendment underlined.)

The amendment of course solved only the immediate problem. Clearly I was now entitled to hear the evidence. It was not so clear that I could make any findings favourable or adverse to the Crown or the Police because the authorization to "comment fully" was still qualified by the limitation against "express[ing] any conclusion of law regarding civil or criminal responsibility." I shall attempt to resolve that problem when I deal with Phase II in this Report.

I therefore now proceed to the task of preparing my Report under the Terms of Reference as interpreted by the Court of Appeal and as amended by the Order-in-Council of May 24, 1984. The original Order-in-Council, the amending Order-in-Council, the Reasons for Judgment of the Court of Appeal, and the Statement of Claim in the action Nelles v. Her Majesty the Queen in Right of Ontario et al. are attached as Appendices 1, 2, 3, and 4 respectively.

B. THE CAUSE OF DEATH

3. STATISTICS

(a) The Wards

As I have said, there were thirty-six deaths (including Kevin Pacsai) on Wards 4A/B during the period June 30, 1980 to March 22, 1981 (hereinafter sometimes referred to as the "epidemic period"). That figure by itself means nothing because hospitals are for sick people and, in the course of things, some sick people must die. But the comparison of this nine-month period with two nine-month periods both before and after produces startling results. Comparison was made by Dr. Anne Gilmour-Bryson, a computer consultant to the Commission, of the periods January to September 1979, October 1979 to June 1980, July 1980 to March 1981 (the epidemic period), April to December 1981, and January to September 1982, of the number of deaths in each period, (a) of patients who had ever been on the cardiology wards, regardless of where in the Hospital they died; and (b) of patients who actually died on the cardiology wards. She prepared charts which are attached as Appendices 5 and 6 respectively.

The chart for total deaths is revealing, showing 64 deaths for the epidemic period against 30, 22, 29, and 28 for the others. The more dramatic difference is, however, found in the chart for ward deaths, where the number of deaths for the relevant period is 34 against figures of 5, 6 (including Laura Woodcock on June 30), 1, and 7. The percentage increase of ward deaths in the relevant period over the average of the other four periods is 625%. The numbers of ward deaths in the other periods bear out the evidence of Dr. Richard Rowe, the Chief of Cardiology at the Hospital, that patients at the Hospital do not normally die on the cardiology wards. Deaths more frequently occur in the Operating Room (O.R.), or in the I.C.U. following surgery, or in the I.C.U. after transfer from the ward. A striking feature of the ward deaths in the period under review was that patients were suffering onset of critical symptoms whose downhill course to cardiac arrest and death was too rapid to permit their transfer to the I.C.U.

(b) The Times

Another startling statistic is found in the time of death. On Wards 4A/B in the relevant period there were twenty-five deaths between midnight and 6:00 a.m. and no more than three in any other six-hour time span. Leaving aside the epidemic period, in all other nine-month periods there was no great variation in the time of death. Even more startling are the figures if we make a comparison among all periods for deaths between 1:00 a.m. and 5:00 a.m. If we look only at that time span, there were twenty-four deaths during the epidemic period and no more than one in the same four-hour time span in any of the other nine-month periods studied. The graphs relating to time are attached as Appendices 7 and 8.

(c) Personnel

Finally, the statistics disclose that the onset of critical symptoms for all of the thirty-six deaths under investigation except one (David Leith) took place when one or more members of a particular nursing team were on duty either on Ward 4A or Ward 4B, and this was so even on the rare occasions when the death occurred at times other than midnight to 6:00 a.m. There were four teams per ward; no one team or team member would normally be on duty for more than 25% of the time. One would therefore expect to see deaths occurring in more or less equal numbers in the presence of all four teams.

(d) Conclusions

These statistics are subject to interpretation but they are not open to question. The Hospital produced data and charts to show that deaths in the Hospital generally were subject to peaks and valleys not coinciding with the epidemic period, but no one disputed the accuracy of the figures set out above. The Hospital's data could only go to justify the lack of awareness by the Hospital Administration of the increase in ward deaths. No statistics were kept of ward-by-ward deaths, and there was nothing remarkable in the total number of deaths in the Hospital during the relevant period. As is pointed out in the Report of the Hospital for Sick Children Review Committee referred to in the Order-in-Council (hereinafter referred to as the "Dubin Report"), the administrative system of mortality review at the time could not and did not bring the increase in ward deaths automatically to the attention of the Administration.

Dr. Rowe and others of the Hospital staff were of the view during the epidemic period that younger and sicker babies were being admitted to the wards which might explain the rash of deaths. An epidemiological study subsequently undertaken by the United States Centers for Disease Control and the Ontario Ministry of Health (the "Atlanta Report") concluded *inter alia* that younger and sicker babies were admitted during that period, but in so doing (as pointed out in a critique of the Atlanta Report (the "Haynes Report") - *infra*), the Atlanta authors had mistakenly included all of the dead babies in a supposedly random sample of the ward population, thus obtaining a false and weighted result. When these data were correctly recalculated in the Haynes Report, *infra*, it was found that there was no significant difference between the epidemic period and other periods regarding age, severity of condition, or prognosis. The Atlanta Report does state that the condition on admittance of those children who died in the epidemic period was less severe (according to Atlanta's classification) than that of patients dying in the other periods, and that conclusion was not challenged.

Hospital doctors were also of the opinion that the increased ward death rate might simply be a result of "clustering", a phenomenon which they felt did take place from time to time for no known reason. No evidence was submitted of any "cluster" remotely resembling what happened in the epidemic period, and the figures for the periods examined by Dr. Gilmour-Bryson do not disclose any support for this theory. Some of the doctors also thought there was a shortage of nursing staff at night which might have contributed to the problem. No actual shortage of nurses at night was documented, however. Some Hospital physicians ascribed the increased death rate to arrival at the Hospital of seriously ill cardiac patients referred there for treatment from Winnipeg. The baby Real Gosselin was the only one of the thirty-six whose deaths are under review who did, in fact, come from Winnipeg.

4. THE DEATHS, THE HOSPITAL, ITS STAFF AND THEIR CONCERNS

The children whose deaths we are considering, together with their ages at times of death, the wards on which they died, the dates of death, the times of the onset of critical symptoms, and the times of death are set out below.

Child	Age	Ward	Date of Death	Time of Onset**	Time of Death
Woodcock, Laura	18 days	4B	June 30/80	0600	0940
Perreault, Alan	25 days	4A	July 8/80	1315	1345
Bilodeau, Andrew	30 days	4A	July 22/80	0125	0210
Taylor, David	14 weeks	4B	July 27/80	0100	0202
Dawson, Amber	11 1/4 months	4A	July 28/80	0130	0240
Hoos, Lillian	15 days	4A	July 31/80	0240	0322
Turner, Philip	28 days	4A	Aug. 1/80	0125	0215
Shrum, Dion	8 weeks	4A	Aug. 9/80	1845	1945
Monteith, Kelly Ann	10 weeks	4A	Aug. 19/80	0330	0445
Murphy, Paul	14 years	4A	Aug. 23/80	2225	2228
Velasquez, Antonio	1 year	4A	Aug. 24/80	0300	0425
Heyworth, Lauretta	11 years	4A	Sept. 2/80	0830	0830
Gage, Brian	29 days	4A	Sept. 25/80	0320	0400
McKeil, Richard	6 1/2 weeks	4A	Oct. 15/80	0345	0427
Adamo, Antonio	9 days	4A	Oct. 19/80	1615	1743
Volk, Francis	12 1/2 weeks	4A	Oct. 23/80	1650	1715
Lutes, Matthew	28 days	4A	Nov. 17/80	2400	0134
Onofre, John	18 days	4B	Dec. 9/80	0320	0410
MacDonald, Darcy	23 weeks	4A	Dec. 13/80	0335	0430
Gosselin, Real	24 days	4A	Dec. 18/80	0215	0316
Lombardo, Stephanie	10 days	4A	Dec. 23/80	0330	0420
Belanger, Jesse	6 weeks	4B	Dec. 28/80	1830	2016
Estrella, Janice	18 weeks	4A	Jan. 11/81	0240	0322
Fazio, Frank	4 1/2 weeks	4A	Feb. 4/81	0330	0445
Floryn, Bruce	19 years	4B	Feb. 7/81	0610	0620
Thomas, Jennifer	9 days	4A	Feb. 12/81	0300	0338
Leith, David	6 weeks	4A	Mar. 6/81	0500	1025
Warner, Colleen	20 weeks	4A	Mar. 7/81	0300	0345
Hines, Jordan	20 days	4B	Mar. 8/81	0410	0643
Gionas, Barbara	6 1/2 weeks	4A	Mar. 9/81	0045	0145
Manojlovich, Michelle	9 months	4B	Mar. 12/81	0230	0335
Pacsai, Kevin*	25 days	4B	Mar. 12/81	0345	1010
Inwood, Kristin	18 days	4B	Mar. 13/81	0200	0300
Gardner, Charlon	21 days	4A	Mar. 18/81	0335	0425
Miller, Allana	1 year	4A	Mar. 21/81	0145	0327
Cook, Justin	14 weeks	4A	Mar. 22/81	0345	0456

* Kevin Pacsai actually died in the I.C.U. but the onset of critical symptoms occurred on Ward 4B.

**For the purposes of this Report, the onset time refers to the time noted in the medical record at which an abrupt and serious change was seen in the child's condition. In most cases, at this time, a Code 23 call went out to summon the immediate attendance of an on-call physician, or a Code 25 call was issued which summoned the arrival of the cardiac arrest team itself.

The organizational structure of the Hospital and its staff have been described in detail in the Dubin Report, and I shall give only the barest outline necessary to make this Report intelligible. Wards 4A/B, as I have said, were adjoining with many common features including a nursing station in the middle. At the time they were, however, separated for nursing duties, each ward having a Head Nurse and four teams of nurses. The Head Nurses generally worked an eight-hour shift, only during the day, and the teams of nurses alternated between long day and long night duty with appropriate time off. Long day service was from 7:15 a.m. until 7:45 p.m., and long night service was from 7:15 p.m. until 7:45 a.m. The half-hour duplication both morning and night was to accommodate the changeover of staff. A team of nurses consisted of a Team Leader who was a registered nurse (R.N.), one or two registered nurses, and one or two registered nursing assistants (R.N.A.s). Generally speaking, a team once formed stayed together and worked together consistently on either Ward 4A or 4B, but from time to time one or more members of a team would be absent or assigned as relief to another team on the other cardiology ward or even on another ward in the Hospital. At any given time (except for the Christmas holiday season), two nursing teams were on duty on the cardiology wards, one team for each ward.

Wards 4A/B were part of the Cardiology Division of the Hospital. The Division was headed by Dr. Richard Rowe and consisted of six other staff cardiologists, and under them a number of cardiac fellows and residents. The cardiologists are the members of staff who are in charge of the medical (as opposed to surgical) treatment of patients with heart problems. The fellows are qualified physicians who are taking advanced studies in cardiology, and the residents are likewise physicians, but are in general pediatrics and less advanced in training in cardiology. The staff cardiologists were permanent members of the Division and were not on duty at the Hospital at night. The cardiology fellows were assigned to several areas in the Hospital where cardiac patients were found (Wards 4A/B, 7G, and the I.C.U.), and were on call at night. Three or four residents were attached to the Division at any given time during the epidemic period and moved every six weeks to another Division in the Hospital.

While the statistics have shown that there were far more deaths over the whole period under review than would ordinarily be expected, it is apparent from the list of deaths that the great clusters were in July - August, and December, 1980, and March, 1981.

In the summer of 1980 some of the nurses on Wards 4A/B became very concerned about the matter, particularly whether some unwitting failure in their nursing care had somehow contributed to the deaths. Their concern was conveyed to the Nursing Administration, as well as to Dr. Rowe who organized in September two "Mortality and Morbidity" Conferences at which cardiologists, fellows, and nurses were present. The first of these was on September 5 at which the deaths of babies Bilodeau, Turner, and Taylor were discussed. The second one was on September 26 when the deaths of babies Shrum, Velasquez, and Monteith were discussed. Generally speaking, the conclusions reached were that the babies died of their clinical conditions and that the nurses had done everything necessary for their care. Some thought was given to the provision of an Intermediate I.C.U. on Wards 4A/B; the Hospital had an I.C.U. where very sick babies were attended to, but it was thought that provision should be made within the cardiac wards for babies requiring more than ordinary attention. Also, the baby Velasquez had been given twice the recommended dose of a usually harmless drug called naloxone immediately prior to his cardiac arrest and subsequent resuscitation effort. For that reason, it was thought appropriate to provide a dosage schedule of drugs to be used in resuscitation.

The doctors were, of course, aware of the deaths. Indeed, every death was discussed in conference the morning after it had occurred, and there were routine reports on autopsies. The doctors themselves were thoroughly convinced the deaths were the result of the severity of the diseases suffered by the babies; the purpose of the Mortality and Morbidity Conferences was to explain the causes of death to the nurses and give them assurance that the increase in deaths was in no way the result of inadequate nursing care. It was intended to have further conferences with the nurses in the fall of 1980, but Dr. Rowe was away and his instructions to that effect were not carried out.

In mid-December 1980, Dr. George Trusler, Director of the Division of Cardiovascular Surgery at the Hospital, wrote to Dr. Rowe expressing his concern about the deaths of patients who had successfully survived surgery and who had thereafter died after leaving the I.C.U., as Dr. Trusler put it: ". . . at a time when we would assume they are out of danger." In consequence, a conference was convened on January 12, 1981. Drs. Rowe and Trusler attended, together with another surgeon and another cardiologist, the Deputy Director of the I.C.U., one of the fellows in cardiology, the Director of Nursing, and several other senior nurses. From the Minutes of that meeting (as varied slightly by Dr. Rowe's testimony), it is apparent that twenty deaths since July 1, 1980, were classified as follows: (Actually there had been twenty-two deaths discussed but two of them are not our concern. Matthew Lutes' death was inadvertently omitted at this meeting. There was also no discussion of Janice Estrella who had died only the day before.)

Terminally ill: Paul Murphy and Lauretta Heyworth.

Expected deaths: Alan Perreault, David Taylor, Philip Turner, and Kelly Ann Monteith.

Unexpected deaths before surgery: Brian Gage, Darcy MacDonald, Real Gosselin, Dion Shrum, and Andrew Bilodeau.

Unexpected deaths after surgery: Antonio Velasquez, Lillian Hoos, Richard McKeil, Stephanie Lombardo, John Onofre, Antonio Adamo, Francis Volk, Jesse Belanger, and Amber Dawson.

Dr. Rowe also testified that the term "unexpected deaths" was intended to refer to all deaths where death was not inevitable.

There was much discussion about the availability of space in the I.C.U. and getting the children to operation or reoperation sooner but the only recommendation was for an Intermediate I.C.U., almost exactly as considered in September. The Minutes of the Meeting were distributed to, among others, the Chief of Pediatrics, Dr. David Carver; this was the first indication to him or to the Hospital Administration of the increased deaths and the concern arising from them. At that time, as the Dubin

Report has told us, the problems seemed to be in hand, and neither the Board of Trustees nor any other Committee of the Hospital was informed.

In none of the meetings was there any discussion of the facts that the deaths were taking place (with a few exceptions) in the early hours of the morning and always in the presence of one or more members of a particular nursing team. Indeed, it is doubtful if any doctor appreciated those facts. The nurses of course did; those on the team were naturally very distressed and some thought was given to breaking up that team to relieve the distress. The other nurses felt only sympathy for the afflicted team. There was no real thought by the nurses or the doctors that the heart drug digoxin or any other medication might have played a part in any of the deaths (other than the baby Velasquez and the drug naloxone). Certainly there was no suspicion of fatal overdose of any drug until Kevin Pacsai died on March 12, 1981. Ironically, on January 11 the day before the conference abovementioned, two post-mortem blood samples were taken from Janice Estrella, one of which, when tested (such tests are referred to sometimes hereafter as "assays") several times between January 12 and January 16, revealed a digoxin level of 72 nanograms per millilitre (ng/ml - one nanogram is equal to one-billionth of a gram); the figure of 72, as we will see, is more than twenty times the highest permitted therapeutic level. Although the laboratory report in the baby's medical record (sometimes hereafter referred to as the "chart") dated January 11, states, "Results flagged were reported today", this level was not reported until January 17 according to the pathologist Dr. Kent Mancer. As disclosed in the Dubin Report, this reading was believed to be produced by an artefact (a contaminant or a digoxin-like substance which is read as digoxin on testing), or to be the result of a mathematical error and was ignored or at least not adequately followed up.

5. DIGOXIN, ITS PROPERTIES

In approaching this subject, I confess at the outset that I am dependent on the opinions of the experts in this developing science. I have been urged to move cautiously and make no findings except where the expert opinion is clear and unanimous. The advice goes against the judicial grain, and I would probably reject it anyway, but more important, I do not find any great diversity of expert opinion, at least on the basics of the drug. There is very little, if any, conflicting evidence on the toxicologic data as they affected any of the children under review. The only conflict is in the interpretation to be placed on the data together with the other relevant facts concerning the symptoms and the deaths of the children. That conflict I will deal with in later chapters. The relatively undisputed facts about digoxin are as follows.

(a) History

Digoxin is a refined form of digitalis which is a derivative of the foxglove plant. It has been used for almost three hundred years in the treatment of congestive heart failure, a condition in which the heart is not effectively pumping blood throughout the body.

(b) Function

Its primary function is twofold. It works to improve efficiency of the contractions (the beating) of the heart so as to assist in the effective circulation of blood from the heart to other parts of the body. It also affects the conduction system of the heart, causing the heart rate to slow, the heart having been forced to speed up to compensate for inadequate pumping. Obviously, if the beat slows too much or ceases entirely from an excess of digoxin, the patient will die.

(c) Pathology

At autopsy there are no detectable physical signs of digoxin whether administered in therapeutic or toxic doses. The only means of detection is by specific tests to determine the presence and concentrations of the drug in the blood or tissues of the patient. Tissue samples for digoxin testing can normally be taken only after death, but blood samples can, of course, be taken either ante-mortem or post-mortem.

(d) Methods of Administration

During the epidemic period, digoxin was available on the wards on open shelves in the medication room. It existed in liquid (elixir) form, in tablets for oral use, and in ampoules of pediatric and adult (for older children) strength for intravenous use. The elixir and the pediatric ampoules were the same strength, but the adult ampoule was five times the concentration and twice the volume, thus containing ten times the amount of digoxin. Records were kept of the amounts distributed to the wards, but these records were destroyed, in the normal course, after three months. There was routine restocking, but if an emergency arose outside of normal Pharmacy hours, digoxin in any form could always be obtained from another ward.

The drug can be administered orally, intravenously, or intramuscularly. The last is painful and seldom resorted to. Digoxin is usually administered orally. In the case of infants, a syringe is used to squirt the elixir into the child's mouth; older children take the drug in tablet form. If a patient is being fed by a naso-gastric tube, the drug may be administered through the tube. Theoretically, the drug could be mixed with food and ingested during feeding but medications are never mixed with food under normal circumstances in a hospital setting. Again, theoretically, the injectable form of the drug could be administered orally to an infant with a syringe or through a naso-gastric tube.

When a child is on intravenous therapy (either for feeding or administering medication), digoxin may be infused by introducing it anywhere along the intravenous line. This method is used by physicians at the Hospital for Sick Children in certain specific situations: under emergency conditions when immediate digoxin therapy is required, when the patient cannot take the drug orally, or when digoxin is being administered for the first time to the patient. When a patient is "digitalized" (first given digoxin), the dose is much larger than the subsequent maintenance doses. This dose is given in three parts: the first (the loading dose) being one-half the total, the second and third each a quarter; thereafter the patient receives maintenance doses, each one-eighth of the total digitalizing dose. At the Hospital for Sick Children, R.N.s are permitted to give many medications, including certain intravenous (I.V.) medications but are permitted to give digoxin only if it is given orally. R.N.A.s were not permitted to administer digoxin, and while they were permitted to administer certain medications orally, in practice they seldom did.

(e) Its Operation

The movement and behaviour of the drug in the body, once administered, depend on the particular method used to administer the drug and, as well, on the individual characteristics of the particular patient. Digoxin has a pharmacologic effect only when it has left the blood and has become bound to tissue - in particular, to heart tissue. If administered intravenously the drug enters the bloodstream directly. If administered orally it enters the blood via the esophagus, intestines (where it is absorbed), and back through the liver. From the blood it is then transported by the circulatory system to various tissues and organs to which it then attaches or "binds" itself. In appropriate quantities it will then have the desired therapeutic effect; in excessive amounts it may produce toxic or even fatal effects either by slowing or by grossly interfering with the rhythm of the heart beat to the point of cardiac arrest.

(f) Its Distribution, the Alpha Phase

The period following administration during which digoxin is distributed to tissues via the circulatory system has been called the "Alpha phase" of distribution.

If the drug is administered intravenously, peak levels or concentrations of the drug in the blood are attained almost immediately. Very quickly the level decreases, and the drug is distributed by the circulatory system to tissues with a "half-life" of 20-60 minutes, that is, half the digoxin administered will be delivered to tissue in the first 20-60 minutes; half the amount then remaining in the bloodstream will be delivered in the next 20-60 minutes, and so on. It takes five half-lives to achieve essentially full distribution of the drug so that it is thought to be attained at somewhere between 1 hour and 40 minutes and 5 hours. Once virtually all the digoxin has been thus distributed to tissues, the low residual concentration which remains in the blood, together with the digoxin which has been released by tissues and is in the process of being eliminated from the body (see infra (g)), establishes a level known as equilibrium or "steady state." Following such distribution of the drug, high concentrations may be found in the heart, kidney, liver, skeletal muscle, and brain. Different tissues absorb different amounts of digoxin; thus different concentrations will be measured in different organs or, indeed, in different parts of the same organ.

If digoxin is administered orally, the peak concentration in blood will occur much later - within 1 to 3 hours after the drug has been ingested. The whole distribution of the drug to tissues is not essentially complete until sometime between 6-12 hours after ingestion. Once again, following such distribution, high concentrations of digoxin may be found in the liver, heart, and kidney.

(g) Its Excretion, the Beta Phase

After "full" distribution (the Alpha phase), digoxin exits slowly from the body. Some elimination of the drug is thought to occur during the Alpha phase but in very small quantities. What happens is that after full distribution, the drug gradually leaves the tissue, re-enters the blood, and is either excreted by the kidneys or broken down by the liver. This has been called the "Beta phase" and has a half-life, depending on many factors (including, for example, the age of the patient, the condition of the patient's kidneys and liver, etc.), which is thought to be between 20 and 80 hours and a concomitant period to complete elimination of between 4 and 17 days. At the end of that time, in theory at least, the Beta phase is complete and most of the dose of digoxin (barring impairment of kidney function) has left the body leaving an amount too small to measure.

(h) Symptoms of Toxicity and Use of Digoxin for Therapeutic Purposes

Just as there are no specific pathologic signs of digoxin, there are no clinical symptoms which specifically indicate digoxin toxicity. If a patient has received too much digoxin, or is suffering an adverse reaction to digoxin (a toxic reaction), it may be manifested by an irregular heart rhythm (arrhythmia), heart block (dissociation between the beats in the upper and lower chambers of the heart), slow heart rate (bradycardia), persistent nausea and vomiting, shallow respirations, sudden cardiac arrest, and perhaps seizure-like activity. These symptoms, either separately or in combination, may be manifestations of digoxin toxicity; they may also be indicative, however, of other cardiac problems.

On the expert evidence before me, it is clear that digoxin has what is described as a "low therapeutic index"; in other words, the concentration of the drug which may produce toxicity does not greatly exceed the concentration which will produce a desired therapeutic effect. It is therefore of great clinical importance in the treatment of any patient, to fix the appropriate amount of the drug which the patient will receive.

To this end, checking of blood levels is a helpful diagnostic tool, but it is very important that the patient's clinical condition be constantly monitored for signs of either inadequate dosage (failure to control congestive heart failure) or excessive dosage (symptoms suggestive of digoxin toxicity).

There are situations in which digoxin should never be prescribed - where the condition of the patient would in fact be adversely affected if digoxin were administered. Digoxin can thus be contra-indicated in a variety of situations where, for example, the patient is more sensitive to the drug than the normal individual or where the patient is already experiencing slow heart rate or particular types of arrhythmias.

6. DIGOXIN, ITS TESTING

(a) History and Acceptable Levels

As noted above, digoxin is a drug which allows only a very small margin for error in dosage. Even a very slightly excessive dose can produce serious toxic effects. Despite the long therapeutic history of digoxin, it was not until the last decade or so that it has been possible to detect digoxin toxicity except by the symptoms displayed, unspecific as they are. In January 1975, at least at the Hospital for Sick Children, it became possible to measure the level of digoxin in the blood of a patient. This enabled clinicians to establish correlations between concentrations of digoxin in blood and the desired therapeutic and undesired toxic effects of digoxin. They could thus regulate the dosage to produce a blood concentration associated with therapeutic effects.

It is clear that toxicity depends on many factors but, generally speaking, the top blood level consistent with therapeutic dosages is considered to be somewhere between 2 and 3 ng/ml. Babies seem to tolerate higher concentrations than adults, and some experts would permit 5 ng/ml in a neonate, that is, an infant less than thirty days old. Most of the doctors at the Hospital for Sick Children, appreciating the danger of the drug, adopted the position that anything over 3.5 ng/ml was potentially toxic; if a higher level was recorded, administration would be withheld and the baby carefully watched for symptoms of toxicity. Dosage would not be resumed until the blood level returned to the general therapeutic range (.5 to 2.5 ng/ml) and then only if no adverse symptoms were manifest. The experts emphasized that higher than usual digoxin levels can be tolerated by children, including infants, without any manifestation of toxicity. On the other hand, symptoms of toxicity can be present although the concentration of the drug would normally be regarded as within the therapeutic range.

(b) The Taking of Tests or Levels

During the epidemic period, there was no routine at the Hospital for the taking of digoxin levels, but antemortem levels were taken upon the request of a clinician. Those requests would be made from time to time to ensure that the prescribed dosage was the correct one and also whenever there was any suspicion of toxicity. Suspicion of toxicity as a cause of death was rarely if ever entertained simply because, as the Mortality and Morbidity Conferences amply demonstrated, the clinical condition of the child was thought, generally speaking, fully to explain the death. The first post-mortem digoxin level test was that of Janice Estrella on January 11, 1981, with the consequences I have described (see ante page 14).

(c) The Methods of Testing

It is very difficult to measure digoxin levels because the quantities in the blood are very small and because digoxin has a high molecular weight. The latter factor means that the drug does not readily lend itself to testing by techniques designed to analyse lower molecular weight substances. As a result of these difficulties, the testing techniques used must, of necessity, be extremely sensitive. There are four techniques in current use as follows.

(i) Radioimmunoassay (RIA)

This involves the use of antibodies, a radioactive digoxin compound, gamma counters, and a computer to determine the amount of digoxin in the patient's blood. The principle is that the patient's digoxin and the radioactive digoxin compete for binding sites on the antibodies, and by use of the gamma counter and computer the amount of digoxin in the patient's blood or serum can be determined (although digoxin tests at the Hospital were usually performed on serum, I shall generally refer to "blood samples" and "blood levels"). The process appears to make an accurate total determination of the amount of the drug present, but the difficulty is that the system tends to include in that total some substances which have a molecular structure sufficiently similar to that of digoxin to enable them to occupy sites on the antibodies used in this assay. The level recorded may thus include digoxin-like substances as well as digoxin and thus give a falsely high reading of the actual digoxin in the blood sample. The process is constantly being improved, however, by the commercial manufacturers who fashion antibodies so as not to attract known similar drugs or substances.

(ii) Fluorescent Polarization Immunoassay (FPIA)

The second method is known as fluorescent polarization immunoassay (FPIA) which is similar to RIA, but fluorescein is used instead of radioactively treated digoxin in the testing procedure. Both the RIA and FPIA techniques use standards and controls containing a known amount of digoxin, a specific digoxin antibody, and a competition process for binding on the antibodies. The FPIA system, however, was not used in the epidemic period but was started in the Hospital for Sick Children in March of 1983 in conjunction with RIA. It has not yet proved to be more specific than RIA; indeed, the results from the two techniques are very similar. The FPIA system is much faster and more convenient and may in time, therefore, replace RIA. The FPIA technique is similar to RIA in one other respect: as it too is an antibody assay technique, other drugs or substances similar to digoxin may occupy binding sites on the antibodies. Levels recorded by use of FPIA, therefore, may also include digoxin-like substances resulting in falsely high readings.

(iii) High Pressure Liquid Chromatography (HPLC)

This is not really a technique for testing because it will not by itself permit the measurement of the actual concentration of digoxin present. What it does is extract digoxin-like substances from the patient's blood prior to another test so that the final test is more likely to measure pure digoxin. By thus removing from the sample substances which might interfere with the RIA and produce a falsely elevated digoxin level, HPLC enables the analyst to conduct, after HPLC, an RIA analysis on the refined sample which will be much more specific in detecting and measuring only digoxin and which will thus produce a much more accurate and reliable result.

(iv) Gas Chromatography and Mass Spectrometry (GC/MS)

This is a combination of two methods known as gas chromatography and mass spectrometry. It involves subjecting a sample to bombardment by a stream of electrons to fragment the molecules and then analyzing the particles so created. The system is valuable in detecting many substances but it is doubtful how useful it is in digoxin testing because of that drug's high molecular weight and because the process is lengthy and expensive. As well, the technique requires high concentrations of the drug to be present to permit analysis. It was used in connection with samples related to Jesse Belanger, Stephanie Lombardo, and Colleen Warner, and I will deal with the results when discussing those babies.

(d) Testing by the Hospital for Sick Children and the Centre of Forensic Sciences

During the epidemic period, the Hospital used the RIA technique exclusively to conduct digoxin tests. At that time, the RIA procedure was the most commonly used technique in clinical and some forensic laboratories for the detection and measurement of digoxin. When the tests involved ante-mortem samples and the levels were found to be in the toxic range, appropriate action was taken where possible by the physicians. The only post-mortem digoxin levels taken by the Hospital during the epidemic period were those for Janice Estrella, Kevin Pacsai, Allana Miller, and Justin Cook. Dr. Graham Ellis of the Hospital undertook experimental digoxin testing on tissue specimens from Jordan Hines and Kevin Pacsai, plus a control sample, on March 20, 1981, using an adaptation of the RIA technique used in testing blood or serum. On March 24-25, 1981, Dr. Ellis reassayed those tissues plus others from Justin Cook. Dr. Ellis considered his results to be unreliable, and the Hospital ceased tissue testing at that time. When the investigation deepened and blood specimens from other children were available for testing, the Centre of Forensic Sciences and, in particular, Mr. George Cimbura (then the head of the Toxicology Section and now the Deputy Director), were approached first to check the Hospital's readings on the blood specimens and secondly to do tests on tissue specimens. Mr. Cimbura was hesitant at first because of lack of experience at the Centre in digoxin testing but undertook the task and devised a system for testing tissue. This involved liquifying and homogenizing the tissue, purifying the sample by an extraction process, and then testing by RIA. In the late summer of 1981 the HPLC technique was also employed. As I have said, the GC/MS technique was also used on specimens related to three babies.

(e) Tissue Samples

Mr. Cimbura and all the other experts concede there are tremendous difficulties in the interpretation of tissue readings. There are three different types of tissue specimens which are of concern: fresh or fresh-frozen tissue specimens, "fixed" or preserved tissue specimens, and exhumed and/or embalmed specimens. There are enormous variations in the distribution of digoxin to tissue from the bloodstream. There are, however, ranges of levels reported in experiments and the published medical and scientific literature that will indicate therapeutic or toxic levels in fresh or fresh-frozen tissue. When it comes to exhumed or embalmed tissue, there can be no quantitative assurance at all (that is, no reliance can reasonably be placed according to all of the experts on the significance of the amount of digoxin actually measured in such specimens, i.e., whether they indicate accurately the concentrations present at the time of death). The degradation of tissue makes any reading quantitatively useless and it is virtually unknown what redistribution of digoxin, if any, takes place in tissues over a long period after death. With respect to fixed or preserved tissue the experts were not so sure. Experiments undertaken by Mr. Cimbura have indicated that for tissue specimens in fixing solutions there are marked reductions in concentration after several months, and Dr. Kauffman (for identification of him and the following experts see _infra_ Chapter 7) was prepared to assume that whatever the level found in fixed tissue, the level at the time of death would have to have been higher. Dr. Spielberg had no faith in fixed tissue readings and Dr. Hastreiter, while accepting the proposition that the levels at death would be higher than those found after fixing in preservative solution, would still be very cautious in interpreting those latter levels.

While fixed or exhumed tissue levels may be of little value quantitatively, it is acknowledged by all experts who testified at the hearings, except Dr. Macklem whose views will be discussed later, that the very presence of digoxin in tissue after death has a qualitative value that is sometimes alarming. Where digoxin had not been prescribed to the individual during life, the presence of digoxin in tissue was generally considered to demonstrate that an unauthorized dose had been administered during the patient's life. Mr. Cimbura's experiments on fixed tissue also give some indication of what the concentration might have been in the fresh tissue. He places no great confidence in their precision, but his estimates (by his opinion and that of others) are conservative and may assist to confirm other evidence of the cause of death.

(f) Substance X

Certain recent research suggests the existence of a digoxin-like substance which may react to the antibodies used in various RIA techniques so as to result in a false-positive reading of digoxin in patients known not to have received the drug. This substance, referred to in evidence before me as "Substance X", has been studied by a number of researchers since early 1983. In experiments conducted by Dr. David Seccombe, of the Shaughnessy and Vancouver General Hospitals in Vancouver, and others, and reported in a letter to the editor of the New England Journal of Medicine in April, 1983, prematurely born children who had not been prescribed digoxin were found to have levels ranging up to 4.1 ng/ml, the highest being that found in a four day old infant. Dr. Seccombe was satisfied that the substance measured was not actually digoxin but some similar substance that reacted to the antibodies used in the various assays. The method used in testing was RIA alone, but seven different RIA "kits" were used, all yielding positive although varying results reflecting the different levels of cross-reactivity between Substance X and the antibodies produced by the different manufacturers. There were no results greater than .2 ng/ml except in premature infants. Similar research has yielded similar results in a number of centres.

Some research at the Hospital for Sick Children also seems to indicate the presence of this substance. On Ward 7F in January, 1982, a most unfortunate sequence of events took place involving a mix-up in medication between epinephrine (adrenaline) and vitamin E as described in Chapter XIII of the Dubin Report. In the course of the investigation, one child not prescribed digoxin was found (by RIA alone) to have digoxin levels of 1.3 ng/ml. In the Dubin Report it is suggested that these levels were the result of a medication error although some doctors at the Hospital do not agree.

From time to time Dr. Steven Soldin of the Hospital did further tests on neonates not prescribed digoxin, either by RIA or FPIA, and did discover levels of a digoxin-like substance up to 1.4 ng/ml. He also experimented on adults using the HPLC, FPIA, and RIA techniques, and while he could not detect any digoxin-like substance in serum, he could in urine, although he could not quantify the amount present. The Hospital has acknowledged that the state of Dr. Soldin's continuing research has not reached the point where it can help us.

Another valuable piece of research for our purposes has come about as a result of the Hospital's decision after the deaths of Allana Miller and Justin Cook to do routine post-mortem tests for digoxin on all patients. This was done under the supervision of Dr. M. James Phillips, Head of the Pathology Department. In 608 autopsies undertaken since March 24, 1981, there were positive readings between 1 and 4.9 ng/ml of digoxin found in post-mortem blood specimens in 97 cases (and 37 in excess of 5 ng/ml, see infra p. 28). In twelve of the 97 cases there was no record of digoxin having been administered at the Hospital for Sick Children. The highest reading in any of those twelve cases was 2.1 ng/ml. None of those tests involved the use of HPLC.

Finally, Mr. Cimbura himself conducted research on the subject. He obtained post-mortem blood samples from 24 children and heart tissue specimens from 20 children not known to be on digoxin and took digoxin levels using both RIA and HPLC. These tests did not disclose any digoxin-like substances above the level of 1 ng/ml (the lowest reading that he considered useful for forensic purposes).

It is quite possible to conclude from this research that the HPLC technique extracts Substance X from the sample and thus precludes its measurement by subsequent RIA analysis. But whether it does or not, the greatest amount of the substance detected in anyone's research to date (4.1 ng/ml in Dr. Seccombe's tests) is miniscule compared with some of the readings we will see to have been encountered in the children whose deaths we are investigating. Substance X, even if it forms part of the digoxin levels recorded, cannot seriously affect their interpretation nor account for levels in the ranges found.

(g) Other Interpretation Problems

Apart from the difficulties in interpreting digoxin levels arising by virtue of the testing techniques used and the nature of the specimen, certain further problems arise attendant upon sampling techniques, storage procedures, etc. The evidence is clear, for example, that blood specimens obtained for ante-mortem digoxin tests must be taken long enough after the last dose of the drug has been given to ensure that the Alpha phase of distribution is complete. Taken prematurely, the level obtained may in fact reflect peak levels resulting shortly after administration and before any significant distribution to tissue has occurred (it is important to remember that digoxin has neither a therapeutic nor a toxic effect until it becomes bound to tissue). In consideration of these difficulties, Hospital policy required that blood samples should be taken at least six hours after administration of the drug, and preferably twelve hours (immediately prior to the next dose). In practice this was not always carried out. Regularly the dose was given at 5:30 a.m. if a digoxin level test had been ordered, with the sample for the test taken as early as 9:00 a.m. that same day.

Similarly, the purity of the sample itself must be assured for any reliance to be placed on the level measured. The integrity of the specimens used to obtain digoxin readings and the appropriateness of the procedures used to store the specimens prior to testing, arise as issues particularly in the cases of Janice Estrella and Kristin Inwood and will be discussed further when I deal with those children.

Quite apart from the effect of death upon digoxin concentrations (see immediately below), it has been suggested that in some situations digoxin unbinds from tissues in abnormally high quantities during life and reenters the blood. If a blood specimen is then taken for testing, it may result in a falsely high reading based on a high concentration of the drug in the bloodstream which is not truly representative of the concentration in tissue. The significance of this phenomenon will be discussed when I consider the death of Kevin Pacsai.

(h) Post-Mortem Factor

There is as well the problem of redistribution of digoxin after death. What happens, it is believed, is that upon death digoxin detaches from tissue and re-enters the circulatory system (or is released from red blood cells into the serum) thus giving an unnaturally high reading that may not be indicative of the concentration of digoxin in the patient's blood or serum immediately prior to death. The experts agree that this occurs sometimes, and they also agree that it does not occur in any uniform way or at any predictable rate; nor does it necessarily occur at all. The experts did not disagree greatly in the range of the increase (the multiplier), setting it somewhere from fractionally over 1 up to 5. The phenomenon is recognized in the literature and, of course, the recent research at the Hospital under Dr. Phillips' supervision is helpful. That research showed, besides the phenomenon with patients not prescribed digoxin (referred to under "Substance X" above), some 37 cases where the reading post-mortem was greater than 5 ng/ml. All of these patients had been prescribed digoxin. In between 21 and 25 of those cases the patient showed recent renal failure which could mean that they had had elevated serum concentrations immediately prior to death as a result of the failure of the body to excrete the drug. (All of the pharmacologists who testified agreed that renal failure or kidney impairment was a clear case where digoxin levels are known to elevate even when appropriate doses of the drug had been given). Except for a reading of "gutter blood" (blood from the pelvic cavity) discussed under Janice Estrella and readings for Gary Murphy discussed under Kevin Pacsai later, the highest readings were two of 12.6 ng/ml, one of which was for a patient with renal failure.

While these readings would certainly support the theory of the multiplier factor, they do not help us much in determining the size of the factor. The difficulty is that while there are in some cases (not all) ante-mortem digoxin levels, in many cases the last ante-mortem level recorded was obtained many days prior to death. We have no way of knowing, therefore, what the level was immediately before death.

Accepting that the multiplier factor does exist, and even taking the highest factor of 5, that factor cannot account for the extremely high blood or serum post-mortem readings with which we are concerned, except possibly those of Kevin Pacsai, and that possibility will be discussed when his death is particularly examined. In the other babies for whom we have post-mortem blood or serum levels, the readings are in the 70s or higher, and a five-fold multiplier would still leave the child with a highly toxic level.

(i) Recent Developments and Conclusions

I think it is safe to say that more has been discovered about digoxin, methods of testing for it, and the interpretation of levels obtained, in the past three years than was discovered in the almost three hundred years that it has been with us as a therapeutic drug. Dr. Seccombe's and Dr. Soldin's research on Substance X continues. Even as the hearings progressed, a conference was held at the Hospital for Sick Children in November, 1983, attended by experts from North America and from elsewhere to discuss the present state of the science. Much remains to be known, however, and to be discovered. In these circumstances, it was suggested that I be cautious in relying on toxicologic evidence. I certainly propose to be cautious in the sense that I shall try not to take any view of, or draw any conclusion from, the toxicologic data beyond those which the weight of expert opinion regards as permissible. It has been suggested by some Counsel that in light of the constant advances in knowledge about digoxin, and about the interpretation of test results, my caution should go so far as to preclude my attaching any weight at all to any data other than those about which there was no expert dispute. I do not think that to be an appropriate attitude for two reasons.

First, a panel of digoxin experts at a meeting held in late March, 1984, has unanimously concluded that the methods of Mr. Cimbura, (i.e., RIA, HPLC followed by RIA) produced the best and most reliable results. The panel was also satisfied that the procedure adopted by him for determinination of the presence of unprescribed digoxin in babies Cook, Hines, Belanger, and Lombardo met the necessary criteria. The panel, while accepting that the GC/MS technique might some day produce more precision, also found that that day is not imminent in the present state of research.

Secondly, I cannot await the research. I am charged to find the cause of death of thirty-six children. Obviously toxicologic information is important. I must accept the best information available, at least if it is not seriously challenged in the present state of the science. I may eventually be proven wrong because the toxicologic evidence upon which I in part based my conclusion may be proved wrong or inadequate. But I shall have done the best I can with the material at hand, and we have received the best material available. That is how I interpret obedience to my mandate.

In the dying days of the hearings, that is towards the close of the evidence in Phase II, an unbridled attack was made on the validity of Mr. Cimbura's methods of testing exhumed tissue by Dr. Peter Macklem of Montreal, a doctor with most impressive credentials, in the course of a speech to the Canadian Society for Clinical Investigation. He used some very extravagant language for which he has since apologized so I will not quote his words directly. He has also acknowledged that at the time of making the speech he was not aware that the results of the tests on exhumed tissue were never offered as proof of an overdose of digoxin, only as proof of the presence of digoxin. Nor was he aware that none of the charges against Susan Nelles was based on readings on exhumed tissue. His basic complaint however remained. He maintained that no exhumation should ever have been undertaken because without controls - that is the exhumation of babies known not to have been on digoxin - the results would have been useless. As I understand it, he considers it basic to research to have a control group as well as an experimental group in order to determine the sensitivity and specificity of the tests used. In particular, the control group if it showed no false positive readings, would lend credence to the positive readings in the experimental group. His concern is that it is not known whether after death either digoxin or some substance similar to digoxin may form in the body.

I am prepared to accept his thesis, but that by no means makes the tests of the Centre of Forensic Sciences valueless. There is no evidence whatever of the formation in the body of digoxin after death. The unanimous expert evidence is that digoxin is not naturally produced in the body during life. Mr. Cimbura's tests at least appear to demonstrate that digoxin is not produced in the body in the period beween death and burial. The difficulties in obtaining the control samples are obvious. Indeed, Dr. Macklem conceded there might be an ethical problem in authorizing exhumations of infants who succumbed naturally for merely control purposes. The alternative method of experimenting on animals - i.e., poisoning and burying them for different periods - also has unattractive features, including delay, and would not necessarily produce results that could properly be applied to human cases. While the tests undertaken may not satisfy the pure scientist, they go a long way to satisfying me as they did the distinguished panel assembled by the Hospital for Sick Children. Dr. Macklem concluded that: "All reasonable people will reject the conclusions of the Attorney-General's Office that exhumed babies were murdered by digitalis overdose because the evidence upon which this conclusion is based is not valid." I do accept the conclusion reached by so many experts that the Centre of Forensic Sciences' tests on tissue of exhumed babies, where the tests resulted in positive readings, determine that those babies received digoxin in life. To paraphrase the words of Asquith L.J. in Candler v. Crane Christmas & Company, [1951] 2 K.B. 164 at 195, if that excludes me from the company of "reasonable persons" I must face that consequence with such fortitude as I can command.

7. THE EXPERTS

When dealing with the facts of ordinary life, the inferences that can be drawn from those facts, and the credibility of witnesses testifying to those facts, a judge can rely on his own experience and knowledge as a citizen, as a lawyer, and as a judge. When it comes, however, to medical and scientific knowledge and theory, he must rely in large part upon the experts. The task is relatively easy when the expert evidence is available and not in serious conflict. When it is not there, or is in conflict, the judge must surmise and choose. It is obviously not the most satisfactory solution but it is the only practicable one.

In this Commission I cannot complain of a dearth of medical and scientific expert evidence. The problem lies in the conflict. To exemplify the problem, I should set forth here the fields of medical and scientific endeavour with which we were concerned and the names of experts whose testimony was received.

(a) Cardiology

This is the science of the heart and its diseases. Wards 4A/B were, of course, the main wards in the Hospital where the diseases of the heart in infants were treated. Among the cardiologists at the Hospital testifying besides Dr. Rowe, were Dr. Rodney S. Fowler, Dr. Robert M. F. Freedom, Dr. Teruo Izukawa, and Dr. Vera Rose. From outside the Hospital we had the evidence of Dr. John E. Fay, an Associate Professor in Medicine and Pediatrics at Queen's University in Kingston who in addition is attached to the Division of Cardiology at the Kingston General Hospital. He was retained by Dr. Ross Bennett, the Chief Coroner of Ontario, in May or June of 1982 to study the charts of the children and give his opinion on the possibility of digoxin toxicity having contributed to their deaths. We had also the evidence of Dr. Alois Rudolf Hastreiter, the Director of the Division of Pediatric Cardiology at the University of Illinois Hospital as well as a Professor of Pediatrics at that university. He was retained as a consultant to the Police and to the Crown in the prosecution of Susan Nelles in late April, 1981. He also reviewed some of the charts - those he was asked to review - and gave his opinion on the cause of death of the children initially in a report dated May 29, 1981, and supplemented on September 30, 1981, and in the summer of 1982, by this time reviewing all of the children. He also assessed the severity of the cardiac ailment of each child on a scale of 1 to 10, the most severe being ranked a 10. Dr. Alexander Nadas (the cardiologist consultant to the Atlanta Report study group - see infra) also rated the children's prognosis for the Atlanta team in three categories: poor, guarded, and good. Dr. Rowe also had three categories: inevitable death (80% or higher), high risk death (40%-80%), and low risk death (0-40%). Three older children considered to be in terminal condition were not ranked by Dr. Rowe.

(b) Pathology

Pathology, of course, involves the study of all diseases but for our purposes the pathologists who testified did so because of their involvement in the post-mortem examination or autopsies of the dead children or in the monitoring of post-mortem digoxin levels after the epidemic period. These pathologists were Dr. M. James Phillips, the Pathologist-in-Chief at the Hospital for Sick Children, Dr. Kent Mancer, Dr. Laurence Becker and Dr. Ernest Cutz, Senior Pathologists, and Dr. Glen Taylor, at the relevant time a resident in Pathology at the Hospital. In addition, Dr. Freedom, besides being a cardiologist, was a Professor of Pathology at the University of Toronto, with a cross-appointment in Pathology at the Hospital, and attended many of the autopsies of those of the children whose deaths were thought to be heart-associated.

(c) Pharmacology

Pharmacology is the study of drugs, their chemistry, and their action. Pharmacokinetics is particularly the study of that action. Pharmacologists are not necessarily medical doctors, but those who testified before me were, and were also doctors of philosophy or the equivalent in their speciality of pharmacology. From the Hospital were the Head of the Division of Clinical Pharmacology, Dr. Stuart MacLeod, and Dr. Stephen Spielberg, a member of that Division. From outside the Hospital came Dr. Ralph Kauffman, now the Director - Division of Clinical

Pharmacology/Toxicology, Children's Hospital of Michigan, and Professor of Pediatrics at Wayne State University School of Medicine, Detroit. He was first retained by the Crown in August, 1982, to advise on whether digoxin had played a part in the deaths. He was given the case summaries from Dr. Hastreiter's report and the medical records of the babies to review. Being a pharmacologist, he, generally speaking, reported on only those babies for whom he had toxicologic data. He did, however, prepare the chart of digoxin "scores" referred to infra under "Epidemiology". Dr. Bernard Mirkin, Director of the Division of Clinical Pharmacology of the University of Minnesota, was retained by the Commission. He, in turn, retained other experts including pediatricians, a cardiologist, and a pharmacologist to assist him.

(d) Biochemistry

Biochemistry is the study of body chemistry and includes the study of blood and other body fluids for the purpose of assisting physicians to come to a diagnosis. In addition to Dr. Seccombe, we also heard testimony from Dr. Graham Ellis and Dr. Steven Soldin of the Hospital for Sick Children. It was Dr. Ellis who was responsible for the tests for digoxin performed at the Hospital by the RIA method during the epidemic period. Dr. Soldin was not so involved at that time but became involved later, particularly in the use of the FPIA method, and as previously noted, in the research into Substance X.

(e) Toxicology

Mr. Cimbura is not a biochemist but a toxicologist. He is a Bachelor of Science in Pharmacy, but his occupation at the relevant time was as a forensic toxicologist, that is, he was engaged in the examination of items for the presence of poisons to assist in the administration of justice.

(f) Epidemiology and the Atlanta Report

Epidemiology is, of course, the study of epidemics. The Atlanta Report referred to earlier was initiated by Dr. David Carver, Chief of Pediatrics of the Hospital, to determine if there had been an epidemic of deaths on the cardiac wards of the Hospital, whether those deaths were related to the administration of digoxin or to other causes, and if the former, how the digoxin was administered. The main authors of the Report were Drs. Clark W. Heath and James W. Buehler for the Centers for Disease Control of Atlanta, Georgia, and Drs. Lesbia F. Smith and Evelyn M. Wallace for the Ontario Ministry of Health. They were assisted in their work by Mr. Robert Kusiak, a medical statistician from the Ontario Ministry of Labour, and by Dr. Madeleine Harris, and Mr. Ronn Andrusco, a statistician and computer systems analyst, both from the Ontario Ministry of Health. They also retained Dr. Alexander Nadas, Chief Emeritus of Cardiology at the Children's Hospital Medical Center in Boston, Dr. Derek deSa, Chief of Pathology at Winnipeg Children's Hospital, and Dr. Kauffman, as consuitants in their specialities. Drs. Buehler, Smith, and Wallace, as well as Mr. Kusiak, gave evidence. Dr. Nadas did not; he declined our invitation, but as I have indicated, we have the benefit of his prognosis for the children and other observations to which I will refer below. A chart summarizing the major findings of the Atlanta Report is attached as Appendix 9.

The "categories" referred to in Appendix 9 are explained in the Report as follows:

> Category A, death with any one of the following criteria: timing of death scored 'unexpected and inconsistent with clinical status' by the consultant cardiologist, mode of death scored 'consistent with special concern' regarding possible digoxin intoxication by the consultant cardiologist, a score of [3 or more] on the 1-5 digoxin scale used by the consultant pharmacologist, or concern expressed by the consultant pathologist that available autopsy findings did not fully explain the cause of death; Category B, deaths with a time of reference between 0000 and 0600 hours and mode of death scored 'consistent' with possible digoxin intoxication but without any of the Category A criteria; Category C, deaths with none of the above criteria.

The "score" referred to in the Report (and included in Appendix 9) is the digoxin score of Dr. Kauffman based upon a complicated set of criteria devised by him. I caution the reader, however, that in the application of the "score" given to any particular patient one does not (as Dr. Kauffman readily admitted) get a scale of probabilities of digoxin poisoning. At most, it gives an indication of the strength of the toxicologic evidence available. There could be no rating higher than "1" unless there were digoxin levels consistent with toxicity or there were measurements of digoxin found where no digoxin was prescribed.

Dr. deSa did not give evidence but his written report to the Atlanta team was received in evidence, and he clarified a part of it in correspondence to us. There were three babies where he: ". . . felt that the findings at autopsy did not explain completely the death of the infant nor the acute mode of death". These babies were Jordan Hines, Kevin Pacsai, and Laura Woodcock.

At the request of the Hospital, Dr. R.B. Haynes and Mr. D.W. Taylor, of the Department of Clinical Epidemiology and Biostatistics at McMaster University's Faculty of Health Sciences, reviewed the Atlanta Report. Their written report, dated December 29, 1983 (referred to earlier as the "Haynes Report"), was critical of the Atlanta Report in a number of respects and, in particular, with respect to the methodology used by the authors of Atlanta. The report was filed as an exhibit before me. Notwithstanding their complaints however, the authors concluded that (at page ii):

> The crude mortality rates reported in Study II of the [Atlanta] Report show a substantial increase in the rate of death during a nine month period (July 1980 - March 1981) in comparison with surrounding periods of time. We conclude that this increase is real and large. The question, then, is not whether there was an unusual increase in mortality during the July 1980 - March 1981 period, but what was the cause of the increase. (Emphasis theirs)

and further at page v. of the Report:

> Furthermore, although each of the individual studies can be criticized from the perspective of epidemiologic methodology, taken together

> they provide convincing evidence that there was, indeed, a substantial increase in cardiology ward mortality that can best be explained by untoward events in the infant room of Ward 4A . . . during the July 1980 - March 1981 period.

(g) Other Experts

In addition to all the experts mentioned above, we had testimony from many other doctors from the Hospital, some with very impressive qualifications. Among these were Dr. Carver (as noted earlier, the current Chief of Pediatrics at the Hospital) and Dr. Harry Bain, his predecessor who, at the request of the Hospital, reviewed the medical charts of the thirty-six children under investigation, amongst others. His initial report, dated June 24, 1982, was amended in November of that year and updated in mid-May, 1983 (the "Bain Report"). He supplemented his report in evidence before me. We also heard from Dr. Marian McGee, Associate Dean, Faculty of Health Sciences, Schools of Medicine, Nursing and Human Kinetics, University of Ottawa, who gave expert evidence on nursing matters and procedures. Other doctors and scientists will be identified where need be in the course of this Report. For convenience, an alphabetical list of doctors and scientists whose expert opinions were received by us in evidence or whose reports were before us, together with their specialities and places of employment at the relevant time, is attached as Appendix 10.

Needless to say, there were many doctors and other experts interviewed whose testimony was not in the end considered necessary.

8. MEDICATION ERROR AND THE THEORY OF ACCIDENT

Medication error is a regrettable fact of hospital life. The problem is dealt with at length in the Dubin Report (Chapter XV, pp. 194 *et seq*.) leading to the recommendation of the adoption (or more properly the speeding-up of the adoption) of the unit dose system. That system, whereby prescribed doses are prepared separately at the Pharmacy and dispensed in ready-to-administer-form, has been found in published medication error studies in the United States to reduce the incidence of medication error by 80% (see the Dubin Report, page 206). However, at the Hospital for Sick Children in the period July, 1980 to March, 1981, the system used involved the preparation of medications on the ward from ward stock - or occasionally borrowed from another ward. Under that procedure, studies conducted in the United States have shown errors in drug administration to occur in anywhere from 5 to 20% of all dosages. (See: *Medication Errors: Causes and Prevention* by Neil M. Davis and Michael R. Cohen, both of Temple University School of Pharmacy, Philadelphia, 1981, and see the Dubin Report, page 194).

That is a most alarming statistic. The Dubin Report says that there is certainly no reason to suspect the Hospital's error rate would be as high as those found in the United States studies, but an incidence even in the lowest range of those reported would be frightening. Fortunately, the published statistics from various hospitals show that very few of the errors are fatal, and most of the errors committed are minor. In the American studies, by far the most common error was the failure to administer the medication at all and many errors were time-related only; underdoses appeared to be more common than overdoses. Nevertheless, there have been shown to be many unreported instances of overdose or administration of the wrong drug which make it necessary to consider whether that could be in part or in whole the explanation here.

First, there has been a study made internally by the Hospital's Department of Pharmacy, again as reported in the Dubin Report. The investigators there examined all Incident Reports in the Hospital (which describe mishaps of many sorts involving patients), and found that there was an average of 18 errors per month out of approximately 5,000 doses per day. Even though the Hospital had a non-punitive reporting system which should encourage the reporting of errors, the investigators found the figures unrealistic, and I think rightly so. With the best will in the world, errors, because not intended, are usually not detected.

We should note here again the most unfortunate incidents of accidental administration of epinephrine for vitamin E resulting in the death of Jonathan Murphy and illness to other children on Ward 7F as reported in Chapter XIII of the Dubin Report. The scientific literature is replete with examples of the accidental confusion of one drug for a like or similarly packaged drug. The cases on Ward 7F are clearly cases in point.

There were, indeed, some incidents of digoxin administration error detected on the cardiac wards and reported in the relevant period. Some of these involved children under investigation here as follows:

(a) Paul Murphy, August 19, 1980. This boy was administered twice his normal dose at 9:00 p.m.

(b) Richard McKeil, September 10, 1980. This baby received his proper dose but after a "hold" order, that is, when digoxin had been prescribed but was to be stopped, possibly because of suspicion of toxicity.

(c) Brian Gage, September 24, 1980. This baby, scheduled to have an administration at 9:00 a.m., was given the same dose as well at 5:30 a.m.

(d) Kristin Inwood, March 12, 1981. This baby was given Kevin Pacsai's prescribed dosage at 5:30 a.m. instead of her own dosage (considerably smaller) scheduled for administration at 9:00 a.m.

In addition, during the epidemic period there were eleven digoxin errors on Wards 4A/B involving children not under review. In one, the correct dosage was given after a "hold" order. In three, the dose was omitted altogether. In one, the correct dosage was given at the wrong time. In one, twice the prescribed dose was given three times before the error was discovered. In five cases, the correct dosage was given once at 5:30 a.m. and inadvertently repeated in the same amount at 9:00 a.m.

We must reasonably presume that many more digoxin errors did occur. It was seriously offered as opinion from some witnesses and submitted in argument from some Counsel that, assuming death by digoxin overdose in any or all of the children under investigation, the overdoses were administered by mistake. There is of course always a chance of fatal error in drug administration, but in my view, if any substantial number of these patients died from an overdose of digoxin, the suggestion that those overdoses were all accidentally administered is preposterous. These errors if they took place must all have befallen one team of nurses, must almost all have taken place in the middle of the night, must all have occurred on these wards only, and must all have been fatal overdoses. It is against all common sense to accept those propositions. No child aside from David Leith died, much less died of digoxin overdose, on Wards 4A/B in the relevant period, except in the presence of one or more members of the same nursing team. The accidental overdose should be less common at night when there is less action and confusion to distract the administrator. The great majority of scheduled administration of medication takes place between 6:00 a.m. and midnight according to the medication sheets in the children's charts. Regular digoxin doses were given at 9:00 a.m. and 9:00 p.m. As we know, most of these children suffered the onset of their critical symptoms in the early hours of the morning. The expert evidence is unanimous that if the digoxin caused the death it must have been administered not more than four hours prior to the onset of critical symptoms. It follows that no error associated with a regularly scheduled digoxin dose could have caused the deaths of any of the children under review whose onset of critical symptoms occurred after 1:00 a.m. and before 9:00 a.m.

The incidence of fatal error in the administration of digoxin is by all accounts very rare indeed. One cannot reasonably believe it would occur so often in so short a period, and almost always only on the long night shift. Indeed, error in the quantity of digoxin administered or in the administration of digoxin for some other drug, while certainly possible, should never happen if the nurses followed the routine imposed by the Hospital which required that all administration of digoxin be double-checked by two nurses as to patient and quantity. Under those instructions, the second nurse was to check the calculation independently and also independently check the amount and type of drug against the medication card and observe the drug being drawn up. The routine for the administering nurse involved ten separate steps, six of which were checking procedures designed to minimize the chances of mistake. As mentioned earlier, registered nurses could administer medications; registered nursing assistants, while permitted to do so, in practice seldom did.

Accidents will happen, of course, even under the best regulated procedures, and they did happen in the administration of digoxin at the Hospital for Sick Children as we have seen, but the theory of multiple, repeated, concentrated, fatal error must be rejected as untenable.

That does not mean, of course, that a particular error may not have occurred and caused the death of a particular child. It was argued that in any case where there was toxicologic evidence either of massive overdose or non-prescribed digoxin, the drug was administered in error. Each case will be examined separately to see if the submission is supportable.

9. THE HEART AND ITS FUNCTION

Before dealing with the individual children and their heart disease, I think it is necessary to say a little about the normal function of the heart. In so doing (and in later describing the illness of each child), I shall try to make the language and the subject intelligible to laymen, of which I assuredly am one, even though that may result in my missing many of the nuances of medical language and the subtleties of the child's condition. I can only hope that knowledgeable readers will be forgiving, and those readers who like me are ignorant will be grateful.

To help in this understanding, I attach as Appendices 11 and 12 respectively, a diagram of the heart showing the principal features and a diagram of the heart showing the circulation. These are, of course, features of a normal heart with normal functions, something denied to most of the children under investigation. I shall try to show the defects in each case by reference to the normal heart.

A normal heart has two double chambers or pumps, the atria and the ventricles, each having a right and a left unit. The right atrium receives the blood from different parts of the body through the veins and passes it through the tricuspid valve to the right ventricle. The ventricle by a pumping action transfers the blood through the pulmonary valve into the pulmonary artery and thence to the lungs. There the blood receives oxygen and returns via the pulmonary veins to the left atrium and through the mitral valve to the left ventricle. From there, it is pumped through the aorta back to the various parts of the body whence it came. The left-side pumping chamber of the heart is the most powerful as it delivers blood around the entire body; it is thus a high pressure chamber while the right side is a low pressure chamber.

Before birth, the lungs are collapsed and very little blood goes through the pulmonary artery into the lungs. During fetal life, most of the blood goes through the ductus arteriosus which is a large channel designed to transport blood from the pulmonary artery directly into the aorta, bypassing the lungs and the left side of the heart. After birth, and after the lungs expand to receive the flow from the pulmonary artery, the ductus constricts and is normally functionally closed within forty-eight hours after delivery.

The heart's electrical system is what creates the beat. The transmitter is the sinus node which is located at the junction of the superior vena cava (SVC - the vein carrying blood from the head, neck, and arms) and the right atrium. From that area, an electrical signal emanates on a regular basis. The signal is transmitted through the atrium and via the atrioventricular node into the two ventricles or pumping chambers. Heart disease may affect the functioning of that electrical system.

The children under investigation all suffered from, or were suspected to have, some defect of the heart or the electrical system, and that is, of course, why they were to be found on the cardiology wards. The particular defect of course had a medical name. When I first describe it, I shall endeavour to explain it in non-medical terms but I shall also attach a glossary of relevant medical terms for convenient reference (see Appendix 13).

10. THE CHILDREN

(A) General

I remind the reader that a list of all the children with some particulars is found on page 10. I am now going to deal with the patients chronologically according to date of death. I shall give their ages at death and I shall describe their illnesses, their courses in the Hospital, their prognoses according to Drs. Rowe, Nadas, and Hastreiter, their terminal events, and where applicable, the findings at autopsy (where an autopsy was conducted). In the course of dealing with each child, I shall also refer to the toxicologic and any other relevant evidence and the opinion of the experts as to the severity of the child's illness and the cause of death.

Much of the experts' opinions stemmed from their perusal of the medical records of the children. These records contained the notes on admission including the past history and present diagnosis, in some cases the treatment record at the referring hospital, the results of catheterization, echocardiogram (procedures to be described later), and other investigative procedures where carried out, the results of surgery and autopsy, as well as notes of the patient's hospital course, including the nursing notes, and records of the medications prescribed and administered. Much of that information was supplemented and tested during the testimony of various witnesses.

The toxicologic data based on tests performed at the Hospital were set out in the chart for the child concerned as well as in certain biochemistry records from the Hospital. The results of the tests performed at the Centre of Forensic Sciences were found in a series of written reports made by Mr. Cimbura to the Crown Attorneys and filed as exhibits. Mr. Cimbura supplemented this information in his oral testimony.

The Final and Preliminary Autopsy Reports generally bear a block-letter heading setting out what would appear to be (and often was) the responsible pathologists' determination of the cause of death. Dr. Becker (a pathologist on staff at the Hospital), stated in evidence, however, that the headings may refer only to the principal disease or disorder. It was accordingly necessary in each case to study the report in full to determine what the pathologists thought to be the specific cause of death.

I invite the reader to refer to Appendix 13, the Glossary of relevant medical terms, when reading the following discussion on each of the children. Although they are dealt with in Appendix 13, there are a number of terms used repeatedly which should now be explained:

(a) Apex

In the context of the discussion which follows, the word "apex" refers to heart rate. According to the evidence before me, a normal heart rate for a child at birth is 140/minute, at six months of age - 110/minute, at one year of age - 100/minute, at three to four years of age - 95/minute, at ten to fifteen years of age - 90/minute, and at fifteen years or more - 75 to 80/minute.

(b) Apnea and Cardiac Monitors

Amongst the monitoring equipment available on the cardiac wards were apnea and cardiac monitors; the former is a machine designed to sound an alarm if the patient stops breathing; it was used on patients suffering from respiratory problems or from apnea, that is, periods where breathing ceases. The latter is a machine which is designed to sound an alarm if an irregularity occurs in the patient's heart rate.

(c) Cardiac Catheterization

This is an investigative technique whereby a small catheter is passed into a vein and through the blood vessels into the heart to determine intracardiac pressure and to detect the existence and type of any cardiac defects or abnormalities. Many of the children whose deaths I am investigating underwent one or more cardiac catheterizations prior to their deaths. For a sick child the procedure is itself dangerous but is often necessary to complete a diagnosis and determine appropriate corrective treatment.

(d) Diuretics

These are drugs administered to cardiac patients to promote the body's excretion of urine. Many of these thirty-six children were prescribed both diuretics and digoxin during the course of their hospitalization. Diuretics include such drugs as furosemide (Lasix), Aldactone, and Aldactazide.

(e) Echocardiogram

This is an investigative technique whereby the heart is examined electronically using sound waves. The procedure assists in identifying structural heart defects or other abnormalities.

(f) Electrocardiogram

This is a study of the heart performed by measuring its electrical activity by means of electrodes placed on the skin. It graphically records heart beat and rhythm.

There are, as well, a few other matters which will be referred to and should now be explained:

(g) Nursing Care

Three levels of nursing care were available to patients on Wards 4A/B: under normal care, a nurse or a registered nursing assistant cared for one to several children - sometimes as many as seven - on a shift. These patients might all be in one room or located in a number of rooms. When "shared care" was ordered, the nurse, usually a registered nurse, looked after one seriously ill and one less ill child in the same room and no others. "Constant care" nursing meant that only one patient was assigned to one registered nurse. It was the policy and the practice on Wards 4A/B that constant nursing care duties were assumed only by registered nurses and not by registered nursing assistants. In both constant nursing care and shared care assignments, the nurse was required to summon a relief nurse to take her place if she left the room on an errand of more than a few seconds or on a regular short or long break. Of all the children whose deaths are under investigation, constant care was in force at the time of death for only Lillian Hoos, Janice Estrella, Bruce Floryn, and Justin Cook. David Leith received constant care from the time of onset of his critical symptoms until his death and shared care prior to the onset.

Shared care was in force at the time of death for Kelly Ann Monteith, Lauretta Heyworth, Michelle Manojlovich, Charlon Gardner, and perhaps Frank Fazio (for whom it was so indicated only on the Tour End Report defined infra).

(h) Resuscitation

If a child's condition changed for the worse to the extent that the nursing staff considered that a physician should be summoned, a "Code 23" was called over the Hospital's public address system; if a child suffered a cardiac arrest or was manifesting symptoms suggestive of imminent cardiac arrest, a "Code 25" was called summoning the arrest team. That team was composed of a cardiac fellow (who, if the cardiac arrest occurred at night, often would be summoned from outside the Hospital), the senior pediatric resident on duty, and medical, surgical, and anesthesiological residents. The registered nurses on the wards would assist the team on its arrival as would nursing supervisors. Generally registered nursing assistants did not participate in resuscitation procedures but rather tended to the other patients on the wards thus freeing the registered nurses to attend at the bedside of the patient in arrest. A "crash cart" containing the drugs and other equipment commonly required for resuscitation procedures was kept ready on each ward and was wheeled to the child's bedside at the time of the Code 25. Digoxin was not listed as one of the medications kept on the cart and, according to all of the Hospital doctors, it was not normally used during resuscitation procedures and should not have been on the crash carts.

Rarely, a "Do Not Resuscitate" order was in place. These orders were made only where death was deemed inevitable and only after consultation with the parents. Such orders were in place for the following children at the time of their deaths - Alan Perreault, Paul Murphy, Lauretta Heyworth, Bruce Floryn, and David Leith.

(i) Tour End Report

This Hospital document was completed by nursing supervisors on the three shifts which they worked (day, afternoon-evening, and night) for each ward which they supervised. On these forms, the supervisors filled in the names and condition of all seriously ill patients, those on shared or constant nursing care, those newly admitted to the Hospital or transferred to the ward, those requiring special treatment, or those with surgery scheduled for the following shift. Brief details of all deaths were noted on the Tour End Report as well. The main importance of this document is that those children whose names appear in the twenty-four hour period prior to death only because they were admitted or transferred at that time, or scheduled for surgery, or whose names were omitted, may be deemed not to have been considered in serious condition. The following children fall into one of the above categories:

Laura Woodcock - not on report.
David Taylor - on report for death only.
Brian Gage - not on report.
Stephanie Lombardo - on report for transfer.

The names of all the other children whose deaths we are investigating appeared on the report accompanied by a note of at least one problem affecting their condition.

(B) Specifics

(1) LAURA WOODCOCK

Diagnosis

The heart defect of this eighteen day old baby girl was a relatively minor one. During life her recognized defect was stenosis (narrowing) of the pulmonary valve, but her circulatory system was close to normal. At autopsy, there was found to be in addition a small ventricular septal defect (a hole in the wall separating the two ventricles) and also a subendocardial infarct (death of an area of heart tissue).

Prognosis

The views of the cardiologists were as follows: Dr. Rowe - lower risk death, Dr. Nadas - good, Dr. Hastreiter - 2 (out of 10) regarding cardiac problem only.

Course of Treatment

The patient was originally treated in another hospital to which she was admitted on June 24, 1980. Her main problem at that time was vomiting. She was jaundiced in appearance, suggestive of a liver problem, and was not gaining weight (was failing to thrive). As well, a lung infection and heart disease were suspected. She was treated with digoxin and antibiotics but digoxin was discontinued almost immediately. Two days after admission to hospital, she was transferred to the Hospital for Sick Children. On admission there she was felt to have mild pulmonary stenosis, but her major problem continued to be her jaundice. She was not felt to be in congestive heart failure. The baby appeared to be stable from June 26-30. Antibiotics were continued, but digoxin was not restarted since her heart problem did not appear to require it. She was to be transferred from the cardiac ward to the gastrointestinal ward.

Terminal Events

In the early morning of June 30 the baby vomited, her blood pressure fell and she seemed lethargic. Her heart rate was irregular but stabilized later. At 6:00 a.m., vomiting occurred again and her pulse became irregular, followed at 9:03 a.m. by cardiac arrest. Her terminal events were marked by complete heart block (total lack of coordination between the upper and lower chambers of the heart) and ventricular fibrillation (rapid uncontrolled contractions of the ventricles). In spite of vigorous measures, she could not be resuscitated. Death was pronounced at 9:40 a.m.

Autopsy

There was an autopsy on this child and the death was reported to the Coroner because of its sudden and unexplained nature. The autopsy revealed extensive pneumonia, and evidence of congenital heart disease as well as other possible causes, but the pathologist was unable to give an explanation for the sudden death of the infant. The Coroner's Investigation Statement, nevertheless, contained the following statement:

> Upon my investigation I was satisfied that death was due to natural causes and determined that no official autopsy or inquest was necessary.

Toxicology

While in the opinion of Dr. Rowe and many of the other cardiologists who testified, the terminal events were consistent with digoxin toxicity, there are no reliable toxicologic data. No ante-mortem readings were obtained. The body was exhumed two years after death and traces of digoxin-like substances were found in muscle tissue, the only tissue specimens tested. The tests were conducted by use of RIA only. The finding is entirely consistent with the digoxin administered to the child prior to her arrival at the Hospital, and by itself, as I have said, means nothing.

The Experts

The experts are certainly not unanimous regarding the cause of death. Dr. Rowe and Dr. Fay said that the pneumonia was enough to cause death. Dr. Hastreiter noted that the pneumonia had existed since birth and, if it had been sufficiently serious to cause her death, would have caused it earlier. Dr. Freedom testified that he thought the major problem was jaundice, but Dr. Hastreiter noted that the jaundice was not getting worse, and both he and and Dr. Mirkin considered her whole liver problem as not severe enough to cause death. In a letter written by Dr. Freedom to the referring physician after the infant's autopsy had been performed, Dr. Freedom explained that autopsy ruled out the serious liver condition thought to have existed. Dr. Nadas, in his report to Atlanta, found the baby's death unexpected, inconsistent with her clinical condition, and consistent with special concern regarding digoxin intoxication. Dr. Rowe considered the ventricular fibrillation surprising but stated that it might have been caused by the infarct seen in the heart on autopsy. Dr. deSa named this child as one of the three where he thought the findings at autopsy did not explain completely the death of the infant nor the acute mode of death. He noted that her congenital heart defect was "relatively minor" and the pneumonitis and liver problem not severe enough to explain the rapid deterioration in the baby's condition. He, like the responsible pathologist at the Hospital, felt there was no definite anatomical cause of death and that the anatomical lesions confirmed at autopsy did not adequately explain the baby's clinical course. Both Drs. Hastreiter and Mirkin thought there was a good probability that the child died of a digoxin overdose.

(2) ALAN PERREAULT

Diagnosis

This twenty-five day old baby boy had a very seriously defective left side of the heart described as "hypoplastic (underdeveloped) left heart syndrome". The left ventricle was extremely small and the mitral valve was essentially closed as was the aortic valve. As a result, blood returning from the lungs was unable to flow out through the aorta to the body. Until the ductus closed, some blood was getting through from the pulmonary artery, but thereafter his condition deteriorated and no surgical intervention was possible (in part because of a hemophilia-like condition preventing even "heroic" surgery). A "Do Not Resuscitate" order was in place.

Prognosis

The views of the cardiologists were as follows: Dr. Rowe - inevitable death, Dr. Nadas - poor, Dr. Hastreiter - 10 (out of 10).

Course of Treatment

This infant was admitted to another hospital on June 25, 1980, at twelve days of age. He was acutely ill and was suffering some respiratory distress at that time. Chest X-rays showed an enlarged heart and signs of congestive heart failure. Digoxin was given, and the baby was transferred to the Hospital for Sick Children later on the same day. During his second week at the Hospital he was reassessed and the initial diagnosis was again confirmed. He was treated with supportive care only. Digoxin was not restarted.

Terminal Events

About 1:15 p.m., July 8, the baby became quite pale, had serious problems with breathing, and a doctor was notified. The baby's cardiac monitor showed only occasional ventricular activity. By 1:45 p.m., he had no respiration, and no apex (heart rate) could be found. As noted earlier, this was one of the babies for whom a "Do Not Resuscitate" order was in place. Therefore, the baby was pronounced dead without any resuscitation effort. The child died peacefully with none of the symptoms of digoxin toxicity.

Autopsy

A partial autopsy conducted on his heart and lungs confirmed the earlier diagnoses and attributed his death to heart failure caused by the severe defects noted above.

Toxicology

Mr. Cimbura received a sample of this baby's serum reported to him as having been taken at autopsy. There is some question of the authenticity of this sample but, in any event, the test for digoxin was negative using RIA plus HPLC and RIA, so there is no toxicologic evidence of poisoning and possibly some positive evidence of the opposite. This was the only serum sample tested, whether ante-mortem or post-mortem, and no tissue specimens were tested. As noted above, while the baby did have some digoxin prescribed and given at the former hospital, none was given to him at the Hospital for Sick Children.

The Experts

Dr. Rowe thought the child had lived longer than expected and none of the other experts expressed any opinion other than natural death. Dr. Nadas, in his report to Atlanta, listed the boy's death as expected and consistent with his clinical condition, but inconsistent with digoxin toxicity.

(3) ANDREW BILODEAU

Diagnosis

This month old baby boy suffered from a defect known as truncus arteriosus (only one large artery leaving the heart). In addition, his pulmonary artery was coming off the aorta instead of the right side of the heart and the common valve for the aorta and the pulmonary artery was thought to be obstructed. As a result, his blood was carried to the lungs at increasingly high pressure which produced progressive heart failure.

Prognosis

The views of the cardiologists were as follows: Dr. Rowe - inevitable death, Dr. Nadas - poor, Dr. Hastreiter - 9 (out of 10).

Course of Treatment

At the time of admission to the Hospital on Friday, July 19, 1980, the baby had already been digitalized and started on furosemide. A cardiac catheterization was to be arranged and a decision regarding surgery made subsequently. Dr. Rowe indicated that the chances of the baby surviving the catheterization procedure were probably quite small, and the prospects for surgical success in the immediate future were poor. Over the weekend of his admission digoxin and diuretics were continued. On the morning of July 21, the baby had an echocardiogram which confirmed the diagnosis of his heart disease and severe congenital heart lesions. On the afternoon and evening of that day, the baby deteriorated with increasingly severe congestive cardiac failure. Despite all measures taken, the baby's condition worsened. He vomited his 9:00 p.m. feeding, and his subsequent feeding and, in consequence, an intravenous was started at 10:30 p.m.

Terminal Events

At 1:25 a.m., July 22, the baby was in respiratory distress, and a doctor was notified by the calling of a Code 23. Two minutes later, a Code 25 was called, since the baby was in severe distress, and his heart rate continued to fall. His terminal events were marked by bradycardia (slow heart rate) and gasping respirations. While there was certainly some deterioration in his condition, the cardiac arrest was sudden. Death was pronounced at 2:10 a.m.

Autopsy

No autopsy was performed as parental consent was denied.

Toxicology

No ante-mortem specimens were tested. His body was exhumed twenty-three months later and various tissue specimens were tested for digoxin by Mr. Cimbura. All tests were conducted using RIA followed by HPLC and RIA. While all test results were positive, and those of most of the brain tissues were in what appeared to be a toxic range, the results were inconclusive because the body had been embalmed and exhumed and digoxin was being administered to him, the last dose a few hours before death.

The Experts

Dr. Rose in her reporting letter to the referring physician described the baby's death as "rather sudden and unexpected" but noted that he had been deteriorating rapidly. Dr. Rowe did not think that he had died sooner than expected but felt that the onset of his terminal events was sudden. He expressed the view that the baby's death was adequately explained by his cardiac condition. Dr. Bain stated that the baby's death occurred a little sooner than expected, but that sudden death was not unusual with this type of heart defect. Most of the experts considered digoxin toxicity unlikely even though they felt his death was a little earlier than expected, but it was nevertheless possible as a cause of death. Dr. Nadas, in his report to Atlanta, rated the baby's death as expected and consistent with both his clinical condition and digoxin toxicity. Drs. Hastreiter and Fay felt there was a very low suspicion of digoxin toxicity. Dr. Fay, indeed, eventually opined that the death was natural.

(4) DAVID TAYLOR

Diagnosis

This three month old baby boy suffered from a reduction in the size of the left ventricle. There were also aortic and mitral stenosis (narrowing of the aortic and mitral valves) and suspected tissue thickening of the inner layer of the left ventricle. In combination, his condition was that of hypoplastic left heart syndrome. The result is to cause back pressure from the left atrium, thence to the lungs, the pulmonary artery, and the right ventricle.

Prognosis

The views of the cardiologists were as follows: Dr. Rowe - high risk death, Dr. Nadas - guarded, Dr. Hastreiter - 8 (out of 10).

Course of Treatment

The baby was admitted to the Hospital on July 25, 1980, for further evaluation of a cardiac murmur, primarily since he was ill-looking and had failed to gain weight in the previous three months. He was digitalized and treated with diuretics and an electrocardiogram was performed, with a plan for a cardiac catheterization in the future. On July 26, the infant appeared to be much better than on admission. His heart and respiratory rates were within normal limits.

Terminal Events

At about midnight, July 26, the baby vomited. At that time, pulse and blood pressure were stable. At about 1:00 a.m., July 27, however, the baby's apex fell to 80 and was irregular. A doctor was notified at once. A few moments later a Code 23 was called. The baby vomited again and the apex was still irregular, at only 70 per minute. At 1:22 a.m., a Code 25 to summon the cardiac arrest team was called and cardiopulmonary resuscitation was commenced. This baby's terminal events were marked by irregular rhythm and heart block, as well as by ventricular fibrillation. The baby could not be resuscitated. Death was pronounced at 2:02 a.m.

Autopsy

At autopsy the child's condition was found to be more serious than had been suspected during life. The heart was grossly enlarged in all chambers with marked over-development of the right ventricle. The suspected diagnosis of endocardial fibroelastosis (thickening of the tissue wall) of the left ventricle was confirmed and described as severe, as was his aortic stenosis. Endocardial fibroelastosis was also reported to exist in the left atrium. The lungs showed marked congestion due to right-sided heart failure. Death was considered to be due to severe heart failure causing both atrial and ventricular rhythm disturbances.

Toxicology

The baby was prescribed and administered digoxin at the Hospital but there were no levels taken of blood or tissue, ante- or post-mortem.

The Experts

There is no doubt that this baby's death was consistent with digoxin toxicity. Dr. Nadas in his report to Atlanta, rated the death as unexpected, consistent with his clinical condition, and consistent with special concern regarding digoxin intoxication. Drs. Hastreiter and Mirkin both leaned to digoxin toxicity as the cause of death, relying in large part on the course of the terminal events. The Hospital doctors, on the other hand, leaned to natural death, although conceding that he had suffered very rapid and sudden terminal events. Dr. Freedom, in reporting to the referring physician, made mention that the infant ". . . unexpectedly sustained a cardiac arrest", but he still did not find the sudden death or the course of the terminal events such as to suspect digoxin toxicity. Dr. Izukawa, the attending cardiologist, had some concern about the baby's death leading him to recheck the prescribed dose and medication record for digoxin and the other medications the baby had received. Since he found both of these to be correct, he did not associate digoxin with the death of this infant. Dr. Rowe considered that ventricular fibrillation was normal in this case because of the abnormal mass of heart muscle.

As noted earlier, at the Mortality and Morbidity Conference held on September 5, 1980, this baby's death was discussed. The results of his electrocardiogram were reviewed and, according to notes made by a nurse in attendance, some question was raised as to whether these results suggested digoxin toxicity. No one who testified who had been in attendance at the meeting was able to elaborate on the discussion, and no one remembered any suggestion being made at the time that digoxin toxicity might have caused or contributed to the baby's death. Dr. Mirkin, however, when testifying before me, indicated that the results of the baby's electrocardiogram taken during his terminal events did suggest digoxin toxicity.

(5) AMBER DAWSON

Diagnosis

This baby girl who died in her twelfth month of life was born with septal defects, one in the wall between the atria, and three in the wall between the ventricles, resulting in the blood pouring from one chamber to the other. She had undergone two operations to correct these defects: one to place a band around the pulmonary artery to prevent the blood pouring from the left side of the heart to the right, and the second to patch the septal defects and then remove the pulmonary band. As a result of the surgery she suffered a paralysis of the diaphragm. She also suffered respiratory problems. The heart operations were successful, but the baby failed to thrive after the second one and was brought back to the Hospital for evaluation five days before her death.

Prognosis

The views of the cardiologists were as follows: Dr. Rowe - lower risk death, Dr. Nadas - guarded, Dr. Hastreiter - 7 (out of 10).

Course of Treatment

The child had been in and out of hospital since birth and digoxin had been prescribed. Following admission to the Hospital on July 23, 1980, her condition remained relatively stable, and the primary emphasis was placed on efforts to increase the baby's nourishment. There was, however, some evidence of early respiratory failure, and she experienced continued breathlessness. Digoxin and treatment with diuretics were continued. She vomited frequently. Twenty-four hours before her death she became very lethargic and again vomited twice. Antibiotics for her respiratory problem were prescribed. No further cardiac problems were noted.

Terminal Events

At 4 3/4 hours before death she was put on I.V. fluids exclusively. She remained stable, but then on July 28 at 1:30 a.m., suddenly exhibited increasing bradycardia, respiratory distress, gagging, and some seizure activity. The resuscitation effort was unsuccessful and she was pronounced dead at 2:40 a.m. The death was unexpected and caused considerable concern among the nurses because of that unexpectedness, but no doubt partly also because the baby was well known to them. The attending physician described her course as one of ". . . sudden, recent deterioration leading to collapse".

Autopsy

As noted above, autopsies are performed routinely at The Hospital for Sick Children - with a parent's consent. In this case the mother not only wanted but prompted a Coroner's Investigation. She asked not only for a Coroner's Investigation but that the autopsy be performed outside the Hospital by pathologists not associated with it. This was very difficult as the Hospital staff perform all children's autopsies in Metropolitan Toronto whether the child died in the Hospital or not. Dr. Bunt, the Coroner, proceeded normally and the autopsy was performed by Hospital pathologists.

That autopsy revealed that the heart surgery was still intact, and the heart repair was in excellent condition although there was evidence of old cardiac injuries resulting from a deficiency of blood flow to the heart. There was evidence of gastromalacia (softening of the stomach wall) and a perforation of the stomach probably precipitated by the vomiting, and evidence of pulmonary collapse but no pneumonitis (inflammation of the lungs). In short, there were many things wrong with the baby which might have contributed to her death, but neither the Final Autopsy Report nor the Coroner's Report could attribute a real anatomical cause of death.

The tragedy of the Coroner's Investigation was that the mother's real concerns were not met. She had had a previous experience of prescribed therapeutic dosages of digoxin administered at home leading to toxicity in the baby and consequent hospital treatment, and remembered specifically saying to Dr. Bunt that she hoped it would not develop that the baby died of an overdose of digoxin. Dr. Bunt does not recall that part of the conversation although he does remember that the mother was concerned about "medication error". I accept the mother's evidence, but obviously her specific concern did not get through to Dr. Bunt because no drug screen, much less a specific test for digoxin, was ordered or taken.

Toxicology

The only ante-mortem blood sample tested for digoxin resulted in a level of 1.9 ng/ml, well within the therapeutic range, on the day after the child's last admission to the Hospital. The post-mortem toxicologic data do not help. The heart and lung tissue of this baby had been fixed in preservative solution. While the Centre of Forensic Sciences' tests showed some digoxin-like substances in the heart on RIA testing alone, and none after HPLC and RIA, the child was receiving digoxin and all traces of digoxin can disappear from fixed tissue after six to nine months. The tissue had been fixed for eighteen months. (Studies conducted by Mr. Cimbura demonstrate that there can be a marked reduction in digoxin levels in fixed tissue in as little as two months after the tissue has been placed in preservative solution.)

The Experts

The Hospital doctors generally believed this to be a natural death concentrating on the respiratory problem, the paralyzed diaphragm, and the perforated stomach. Dr. Rowe admitted that there was still doubt about the direct cause of her death but favoured the view that her stomach perforation may have been sufficient to trigger her cardiac arrest. Dr. Bain was certain that the perforation of the stomach caused the cardiac arrest. Dr. Rose attributed death to the child's multiple problems including hypoxia (lack of oxygen) and sepsis (infection). Dr. Fay would put suspicion of digoxin overdose as slight, but Dr. Hastreiter thought the probability of digoxin toxicity as a cause of death was fair. Dr. Mirkin, while conceding that the cause of death was unclear, did not think digoxin toxicity was the cause. Dr. Nadas, in his report to Atlanta, rated the baby's death as unexpected and inconsistent with her clinical condition. He also considered that the terminal events were consistent with digoxin toxicity.

While most of the doctors were of the opinion that the terminal events were consistent with digoxin toxicity, they equally found them consistent with other natural causes. Although the child was very sick she was not considered at imminent risk, and the death in that sense was unexpected.

(6) LILLIAN HOOS

Diagnosis

This two week old baby girl suffered from hypoplastic right heart syndrome including a defect known as "pulmonary atresia" (the pulmonary valve leading from the right ventricle to the pulmonary artery was closed), and the right ventricle was much reduced in size. The blood that comes to the right side is forced through the foramen ovale (the opening between the atria that exists before birth and usually closes after birth), and thence into the aorta, but in the process is not oxygenated by the lungs. The baby's ductus arteriosus remained patent (open) which may in this instance have had a beneficial effect.

Prognosis

The views of the cardiologists were as follows: Dr. Rowe - high risk death, Dr. Nadas - poor, Dr. Hastreiter - 8 (out of 10).

Course of Treatment

The only known solution to this baby's cardiac problem is to create an artificial ductus or channel from the aorta to the pulmonary artery. This procedure, known as a "Waterston Shunt" operation, was duly performed two days after birth. It is a somewhat desperate operation and the long-term prognosis is poor. The baby survived the operation but had a difficult post-operative course. She was transferred to the wards from the I.C.U. on July 23, 1980, at seven days of age, in relatively stable condition. She continued to have episodes of heart failure which improved with medical treatment including prescribed doses of digoxin, diuretics, and fluid restriction. By July 29, some thirty-six hours before her death, her respiratory difficulties had increased; she was experiencing quick shallow breathing patterns, and it was thought that her shunt may not have been functioning properly. She was placed on constant nursing care and on an apnea monitor to observe her breathing.

Terminal Events

Although the baby was in relatively stable condition before, with her vital signs stable, her heart rate regular, and her respirations satisfactory, she suffered a sudden attack of extreme bradycardia, respiratory distress, and cardiac arrest on July 31 at 2:40 a.m., from which she could not be resuscitated. Death was pronounced at 3:22 a.m.

Autopsy

At autopsy the clinical diagnoses were confirmed and it was noted that the shunt repair was intact and functioning. Both lungs were seen to be collapsed. No specific cause of death was given although the many problems known to exist were noted. "Hypoplastic right heart syndrome" was listed as the heading of the Final Autopsy Report.

Toxicology

There was a digoxin reading of 1.7 ng/ml on July 29, but no post-mortem readings on blood or tissue.

The Experts

The experts agreed that the onset of her terminal events was sudden and her death was unexpected as to time, but it certainly could be explained by her condition. Equally it could have been caused by digoxin toxicity. Dr. Rowe believed the death to be the result of the child's clinical condition, specifically, her severe heart malformations in addition to her respiratory difficulties. Dr. Nadas, in his report to Atlanta, rated this baby's death unexpected but consistent with both her clinical condition and digoxin toxicity. Dr. Fay said there was only a low level of suspicion, while Dr. Hastreiter thought there was a fair probability of digoxin toxicity.

(7) PHILIP TURNER

Diagnosis

This four week old baby boy's defects included an incompletely developed left heart. Both the left chambers and the aorta were smaller than normal, and the latter was obstructed. In addition, he had atrial and ventricular septal defects and a defect in the mitral valve which was not discovered until autopsy. In essence he suffered from complete abnormality of a whole series of areas in the left region of the heart and in the aorta.

Prognosis

The views of the cardiologists were as follows: Dr. Rowe - inevitable death, Dr. Nadas - poor, Dr. Hastreiter - 9 (out of 10).

Course of Treatment

The baby was born on July 4, 1980, and was admitted to the Hospital on July 17. Two days later he underwent corrective surgery on the aorta and ligation of the patent ductus arteriosus (surgical closing of the ductus). After the operation he went to the I.C.U. where he suffered partial lung collapse and breathing difficulties. Nevertheless, he was considered sufficiently improved to return to the ward after eleven days. According to Dr. Rowe, the baby seemed to be improving. The very next day, however, July 31, a lung X-ray revealed complete collapse of the left lung. He was receiving digoxin therapy intermittently as prescribed until the day of his death.

Terminal Events

After two days on the ward, while relatively stable although suffering from hypothermia (low temperature), he suffered sudden episodes of bradycardia and arrhythmia at 1:25 a.m. on August 1, followed by cardiac arrest. Resuscitation efforts were unsuccessful. Death was pronounced at 2:15 a.m.

(PHILIP TURNER)

Autopsy

Besides noting the cardiac defects referred to above, the autopsy revealed that the baby also suffered from ischemic encephalopathy (brain damage arising from the deficiency of blood). Death was attributed to congenital heart disease complicated by that brain damage.

Toxicology

There were some digoxin problems during life, and several doses were ordered held, but all the readings were within therapeutic levels. There are no toxicologic post-mortem data.

The Experts

There is no doubt whatever that the baby was very sick and his prognosis was poor, but there was some hope that the surgery had at least temporarily resolved the situation. Indeed a report from the Hospital to the baby's referring physician written six days prior to his death said: "Presently the child looks good in the Intensive Care Unit and we are hoping for a pleasant satisfactory result". All the Hospital doctors were satisfied that his death was caused by his anatomical condition, although Dr. Izukawa had considered the baby's heart failure to be under control prior to the onset of critical symptoms. They conceded that the terminal events were consistent with digoxin toxicity although no thought was given to it at the time.

Dr. Fay had only a little suspicion of digoxin toxicity while Dr. Hastreiter was more concerned about the possibility. Dr. Nadas, in his report to Atlanta, rated the baby's death as unexpected but consistent with his clinical condition. He also found the death consistent with special concern regarding digoxin toxicity. Dr. Mirkin did not think digoxin toxicity caused the infant's death but, contemplating his symptoms, considered the possibility of digoxin toxicity during life, notwithstanding the fact that his ante-mortem levels were within the therapeutic range.

(8) DION SHRUM

Diagnosis

The basic problem with this eight week old baby boy's heart was that the veins from the lungs instead of returning the oxygenated blood to the left side of the heart returned it to the coronary sinus (a vein between the upper and lower chambers of the heart) which in turn drained into the right atrium. This meant that the oxygenated blood from the lungs mixed with the blue blood from the body in the right side of the heart. There was an atrial septal defect, so some of this mixed blood did get to the left side, but what went into the aorta was mixed and not oxygenated blood.

The effect of this transposition was that the right side of the heart increased in size, and the blood kept coming back to that side and going back out to the lungs with only a small portion passing through the septal defect to the left side and thence to the aorta. As a result, the right side of the heart can begin to fail as the right ventricle becomes overloaded with too much blood. Left unattended, such a condition leads to progressive heart failure and death.

Prognosis

The views of the cardiologists were as follows: Dr. Rowe - high risk death, Dr. Nadas - guarded, Dr. Hastreiter - 7 (out of 10).

Course of Treatment

The baby was admitted to the Hospital because of tachypnea (rapid breathing), irritability, and cyanosis (blue colour indicating a lack of oxygen in the blood). On arrival he was experiencing a severe degree of heart failure and, as a result, had a very enlarged liver. He was started on digitalizing doses of digoxin and on diuretics. The day after admission, on August 9, 1980, he was catheterized. As I have said, this procedure is in itself dangerous to a sick baby but is necessary to complete the diagnosis and determine the corrective treatment. The child tolerated the procedure well and was returned to the ward.

(DION SHRUM)

Terminal Events

Back on the ward the child was closely monitored. About 5:30 p.m. that same day, a doctor was notified because of a deterioration in the baby's condition. The I.C.U. resident attended to determine whether the infant should be transferred to the I.C.U. but felt that he could be satisfactorily cared for on the ward if frequent monitoring was continued. At approximately 6:45 p.m. the baby went into severe distress and developed a very irregular heart rate, as well as seizure-like activity. By 7:00 p.m. he had become bradycardic and developed complete heart block with varying ventricular heart rate followed by cardiac arrest. He was unable to be resuscitated and was pronounced dead at 7:45 p.m.

Autopsy

The autopsy confirmed the diagnosis referred to above and noted the pathologic effects of that condition. Death was attributed to heart failure secondary to total anomalous pulmonary venous drainage to the coronary sinus.

Toxicology

The baby was prescribed digoxin but received only part of his digitalizing dose. An ante-mortem level was ordered but the child did not survive long enough for the test to be conducted. No post-mortem tests were performed.

The Experts

The Hospital doctors classified this death as natural. Dr. Nadas, in his report to Atlanta, rated the infant's death as unexpected but consistent with both his clinical condition and digoxin toxicity. Since his death did not occur between midnight and 6:00 a.m., and since no Category A criteria were present, the Atlanta Report placed this death in the Category C or natural death category. Dr. Fay originally thought the death suspicious of digoxin toxicity, but seemed inclined when testifying to label it as natural. Dr. Hastreiter originally considered there was a good probability of digoxin toxicity noting that both the heart block occurring hours after a catheterization, as well as the timing of the arrhythmias, were unusual. In testimony, he changed his opinion to fair probability of digoxin overdose.

(9) KELLY ANN MONTEITH

Diagnosis

The problem with this ten week old baby girl's heart was that the left coronary artery designed to carry blood to the muscle of the left ventricle rose not from the aorta as is normally the case but from the pulmonary artery. With this condition, when the pressure in the pulmonary artery falls (after birth), no blood passes through the coronary artery, and the left ventricular muscle ceases to be supplied with oxygen. The right coronary artery expands, and some oxygenated blood does pass to the left ventricular muscle through the small vessels uniting the two systems, but inevitably the left muscle suffers and dies. Furthermore, the mitral valve is damaged from lack of oxygen producing massive enlargement of the left atrium which in turn may compress (as it did in this case) the main respiratory passage to the left lung. As a result, early breathing difficulties develop requiring treatment and surgery to transpose the coronary artery back to the aorta from the pulmonary artery.

Prognosis

The views of the cardiologists were as follows: Dr. Rowe - high risk death, Dr. Nadas - poor, Dr. Hastreiter - 9 (out of 10).

Course of Treatment

The infant was admitted to the Hospital on August 14 and spent one day on the ward before transfer to the I.C.U. that afternoon, returning to the ward on August 15. The success of surgical treatment to correct this type of defect depends on just how much of the heart muscle has died. The survival rate is small, but after catheterization to confirm the diagnosis on August 18, 1980 (which the baby apparently tolerated well), surgery was scheduled for August 21. The baby's progress following catheterization was reasonably good. Vital signs were stable although respiration was laboured. She was prescribed and received digoxin and diuretics.

(KELLY ANN MONTEITH)

Terminal Events

The baby died before surgery could be attempted. On August 19, early in the morning, and some seventeen hours after the catheterization, at about 3:30 a.m., the baby vomited, stiffened, became limp, and then went into cardiac arrest. She suffered ventricular fibrillation which was temporarily converted (i.e., to normal rhythm), and then went back into ventricular fibrillation and experienced bradycardia. Resuscitation was unsuccessful and she was pronounced dead at 4:45 a.m.

Autopsy

The autopsy confirmed the diagnosis and found severe damage to the heart from lack of oxygen.

Toxicology

The baby's last dose of digoxin was given at 9:00 p.m. August 18. A level of 2.5 ng/ml was found in a test taken earlier that day which may or may not have been reported to the clinicians before her death. In any event, the reading was within the therapeutic range, and Dr. Freedom in evidence said he was not concerned. No post-mortem tests were undertaken.

The Experts

All the experts agreed the prognosis for the baby, even of her surviving surgery, was very poor. Dr. Freedom put the risk of death at 100%. Drs. Rowe and Bain were equally pessimistic, though prior to autopsy Dr. Rowe did not consider the baby to have been at imminent risk at the time when she died. He also stated that ventricular fibrillation was normal with her particular type of cardiac abnormality. All the Hospital doctors found the terminal events sudden but believed her death to be due to her clinical condition. Dr. Nadas, in his report to Atlanta, found the baby's death expected and consistent with both her clinical condition and digoxin toxicity. Dr. Hastreiter testified that a digoxin overdose was "possible".

(10) PAUL MURPHY

Diagnosis

This boy was nearly fifteen years old when he died. He had been in and out of hospital throughout his life suffering from a very large ventricular septal defect with pulmonary atresia (no opening to the pulmonary artery). His condition is also described as "tetralogy of Fallot", the "tetralogy" meaning a collection of four things being: the septal defect, the pulmonary stenosis, the dextral position of the aorta (displacement to the right), and right ventricular hypertrophy (overdevelopment). His condition was particularly complicated because the aorta, instead of curving to the left side of the body, curved to the right. The aorta became very large because it was carrying most of the blood and was compressing the superior vena cava. He had earlier had the aorta removed and a plastic tube graft substituted for it to relieve that pressure. He had also had a patch placed on the pulmonary artery to enlarge it. Despite these efforts, there was never enough blood going to the lungs, and he was continually short of oxygen with resultant ventricular failure. At the time of death no further surgical intervention was possible, and he was in intractable congestive heart failure. His last admittance to the Hospital was to see if an adjustment to his medications might assist, and also for neurological evaluation.

Prognosis

The views of the cardiologists were as follows: Dr. Rowe - excluded from list, Dr. Nadas - poor, Dr. Hastreiter - 9 (out of 10).

Course of Treatment

He was last admitted on August 19, 1980, and suffered a gradual deterioration of his condition of congestive heart failure. His medical treatment aimed primarily to make the boy more comfortable. He was not on intravenous feeding or intravenous medication.

Terminal Events

The boy's death according to all the experts was inevitable. There was, however, a sudden arrest and death was pronounced at 10:28 p.m. on August 23. No attempt was made to resuscitate him, a "Do Not Resuscitate" order having been in place. Except for the sudden onset of his terminal events, he did not manifest any of the usual symptoms consistent with digoxin toxicity, namely, documented arrhythmias, ventricular fibrillation, bradycardia, vomiting, and the like.

Autopsy

Parental permission was not granted for an autopsy.

Toxicology

The child was on digoxin but there were no post-mortem tests undertaken either on blood or tissue. A recent ante-mortem test produced a level within the therapeutic range.

The Experts

All the Hospital doctors and Dr. Fay concluded the death was natural. Dr. Nadas, in his report to Atlanta, rated the boy's death expected, consistent with his clinical condition, and inconsistent with digoxin toxicity. Drs. Fowler, Rowe, and Freedom, while ascribing the death to natural causes, thought it consistent with digoxin toxicity primarily because of the suddenness of the terminal events. Dr. Hastreiter was originally concerned by the suddenness of the terminal events but later concluded the boy died a natural death.

(11) ANTONIO VELASQUEZ

Diagnosis

This one year old baby boy, like Paul Murphy, had tetralogy of Fallot, though in a less serious form: specifically and most prominently, he had a large ventricular septal defect and an obstructed pulmonary artery. In this condition, the venous blood comes from the vena cava through the tricuspid valve in the normal fashion but then has a choice either to proceed normally into the pulmonary artery or to proceed abnormally through the septal defect into the left ventricle. Because the pulmonary artery is small, there is an obstruction to the first route and a tendency for the blood to proceed predominantly on the abnormal route. Thus the blue blood becomes mixed with the red, and thus the "blue baby condition" is produced. The baby was brought from his native St. Lucia to the Hospital for palliative shunt surgery to be followed when he was older by further corrective surgery.

Prognosis

The views of the cardiologists were as follows: Dr. Rowe - lower risk death, Dr. Nadas - good, Dr. Hastreiter - 4 (out of 10).

Course of Treatment

There are two surgical treatments available. The eventual treatment has to be the repair of the septal defect and the opening of the pulmonary artery, but the initial palliative surgery can be performed by way of a Blalock-Taussig shunt (i.e., a tube is inserted linking the artery going to the right arm with the right pulmonary artery), thus shunting some of the aortic blood into the pulmonary artery and relieving some of the "blueness". He was admitted on August 19, 1980, and on August 21 the operation was performed successfully. He was returned to the ward on August 22 for recovery and eventual return to his homeland. His progress was normal. He received prescribed doses of digoxin on the first two days following admission after which it was discontinued. On August 23 the baby developed fever and tachycardia (rapid heart rate) and infection was feared which might adversely affect recovery from the shunt operation. Because of his discomfort, codeine had been administered on August 22, and was given throughout the day on August 23 to relieve the problem.

Terminal Events

Throughout the evening on August 23 he remained in stable condition although until midnight he suffered from tachycardia and fever. After that time his vital signs were regular, and he remained on a monitor for observation. Early in the morning of August 24, he developed bradycardia and became unresponsive. The symptoms were attributed to the effects of the codeine, and naloxone, an antidote to that drug, was given. In error, the dose of naloxone was twice that recommended. The child initially responded favourably but he was not fully responsive, and a second dose of naloxone (again twice that recommended) was given five minutes later. (This drug is frequently administered at five-minute intervals until a satisfactory result is obtained.) Almost immediately after the second dose, at 3:20 a.m., he suffered a cardiac arrest and could not be resuscitated. Death was pronounced at 4:25 a.m.

Autopsy

The doctors were very concerned about the death because it was quite contrary to expectation. Some concern was initially expressed about the overdoses of naloxone, but the drug has a wide safety margin, and it was not felt that the size of the doses was likely to have caused injury. In any event, the death was reported to the Coroner, and an autopsy was held under his auspices. The autopsy confirmed the diagnosis of tetralogy of Fallot and disclosed a fully operative shunt. There was no sign of infection. The Coroner listed the cause of death as "undetermined" and indicated that although naloxone had not been detected by toxicology, possible death due to an overdose of this drug could not be ruled out. Codeine was discovered in the blood by tests at the Centre of Forensic Sciences at 0.005 mg% which was reported as ". . . within the normal therapeutic range for an adult." The Final Autopsy Report stated: "Post-mortem codeine level within normal therapeutic range."

(ANTONIO VELASQUEZ)

Toxicology

No digoxin levels ante-mortem or post-mortem were obtained.

The Experts

There is no doubt that this child's death was regarded by all experts as sudden and surprising. Although none of the Hospital doctors had ever heard of a fatal reaction to an overdose of naloxone, there was no other apparent cause of death and they accordingly accepted the explanation that the death was the result of an idiosyncratic reaction to the drug. It now appears from the literature that there have been two recorded cases of older patients suffering an adverse reaction to naloxone, one of which proved fatal. Nevertheless, adverse consequences are very rare indeed.

While all doctors considered the death unexpected and inconsistent with the baby's clinical condition, there was no such unanimity when considering the relationship of the death to digoxin toxicity. Dr. Bain considered the death unexplained and stated that there might be some relationship to codeine and naloxone. Dr. Nadas, in his report to Atlanta, rated the boy's death unexpected, inconsistent with his clinical condition, and consistent with special concern for digoxin toxicity. Dr. Fay thought digoxin toxicity possible, but he thought that codeine was the more likely cause of the bradycardia. Dr. Hastreiter thought either codeine or digoxin could have caused the bradycardia although he felt there was a good probability of digoxin overdose. Dr. Mirkin considered the possibility of death from digoxin toxicity low. Dr. Kauffman expressed the view that naloxone as the cause of death was unlikely but, in the absence of toxicologic data concerning digoxin, he was unable to express an opinion as to the possible involvement of digoxin in the death.

(12) LAURETTA HEYWORTH

Diagnosis

This eleven year old girl (whose first name is sometimes written "Laurette") was born with a normal heart. She had two related major congenital problems: myelomeningocele (protrusion of the spinal cord through a defect in the spinal column) and hydrocephalus (a condition producing accumulation of fluid in the skull, which if untreated may cause the brain to atrophy or waste). She had been given a ventricular atrial shunt from the brain to the right atrium in an effort to drain away the excess fluid. By 1979, she had developed multiple clots obstructing the flow of blood in the lungs. As a result, the shunt was removed and replaced, but the damage to her lung vessels remained permanent. She then experienced thickening of the wall of the right ventricle and developed right heart failure. She was also suffering from rheumatoid arthritis. Eventually her heart failure became intractable, and she was dying at the time of her last admission to the Hospital. There was a "Do Not Resuscitate" order in place.

Prognosis

The views of the cardiologists were as follows: Dr. Rowe - excluded from list, Dr. Nadas - poor, Dr. Hastreiter - 9 (out of 10).

Course of Treatment

This child was admitted to the Hospital on August 26, 1980, because of abdominal pain plus an accumulation of fluid in the stomach and gross heart failure. She was treated with large doses of diuretics which in turn led to difficulties with the sodium levels in her blood. She was also treated with digoxin. On August 28 she vomited several times and was taken off oral feedings. Over the next several days she became increasingly cyanotic and experienced swelling of the abdomen, although she was again able to tolerate fluids.

Terminal events

On the night of September 1, the child was in great pain, and little could be done to alleviate it. She developed irregular heart beats and shortness of breath. In the morning of September 2, at 8:30 a.m., she suffered ectopic (misplaced) heartbeats and suddenly died.

Autopsy

There was no autopsy; parental consent was denied.

Toxicology

The last digoxin reading was 2.5 ng/ml on August 27. Digoxin toxicity was suspected and the drug was held. It was reordered on August 28. No further readings, ante-mortem or post-mortem, were obtained.

The Experts

The experts were virtually unanimous that the death was a result of her many anatomical problems. Only Dr. Hastreiter felt that there was any likelihood of digoxin toxicity, and he eventually reduced that to a small probability. All the others, despite the suddenness of the terminal events, could see no such realistic possibility. Indeed, Dr. Nadas, in his report to Atlanta, found the girl's death expected, consistent with her clinical condition, and inconsistent with possible digoxin toxicity.

(13) BRIAN GAGE

Diagnosis

This month old baby boy had a complete transposition of the main arteries of the heart, that is, the pulmonary artery came off the left ventricle, and the aorta off the right. In addition, the baby had a ventricular septal defect. As a result, the baby's pattern of blood circulation was grossly distorted. The blood from the lungs went back and forth to and from the left ventricle, and the blood to the rest of the body came to the right ventricle and was pumped back to whence it came. The blue blood could only reach the lungs via the foramen ovale or the ductus arteriosus. In these cases, when the ductus shuts, the baby will die from progressive lack of oxygen.

Prognosis

The views of the cardiologists were as follows: Dr. Rowe - lower risk death, Dr. Nadas - guarded, Dr. Hastreiter - 6 (out of 10).

Course of Treatment

The long-term correction for this condition is to switch the arteries, but in the short-term, one can only enlarge the foramen ovale to allow more blood to pass through until the baby is old enough to tolerate the major surgery. On admission to the Hospital on September 5, 1980, he was cyanotic, had an enlarged chest, and was breathing rapidly and shallowly. He had a cardiac catheterization that night, but prior thereto suffered an episode of bradycardia from which he was successfully resuscitated. The baby remained in continuing heart failure and continued to lose weight although vital signs were stable. He was digitalized and treated with diuretics. He experienced renal difficulty caused by an episode of acute tubular necrosis (death of kidney tissue).

Terminal Events

The surgery to enlarge the foramen ovale was scheduled for September 25, but on the same morning, at 3:20 a.m., he developed bradycardia and lowered respirations, and went into cardiac arrest from which he could not be resuscitated. Death was pronounced at 4:00 a.m.

Autopsy

At autopsy, the cardiac diagnoses mentioned above were confirmed. Lung and liver congestion showed the results of heart failure. No precise cause of death was given. The heading of the Final Autopsy Report is only "transposition of great vessels".

Toxicology

This is a baby who received an accidental extra dose of digoxin in the early morning of the day before death leading to the taking of a digoxin level later that day which was 3.5 ng/ml. Although the evening dosage was withheld, the reading was not considered disturbing. The baby was exhumed twenty-one months later and some exhumed tissue specimens were tested, but the results were inconclusive.

The Experts

Dr. Freedom in his reporting letter to the referring physician stated: "It is unclear as to the precise cause of death, but most likely it was due to a hypoxic episode." In addition to hypoxia (shortness of oxygen), he thought that renal (kidney) failure might have contributed to the death. Dr. Rowe felt that the baby's terminal events could have been caused by his natural deterioration from severe congenital heart disease although the events were equally consistent with digoxin toxicity. Dr. Nadas, in his report to Atlanta, rated the baby's death unexpected and consistent with both his clinical condition and digoxin toxicity. Dr. Fay had little suspicion of digoxin toxicity, but Dr. Hastreiter considerably more. He stated that in his opinion the baby had recovered from the effects of the tubular necrosis and that surgery would normally be expected to succeed in the correction of the baby's cardiac problem. Dr. Mirkin thought the baby's condition was deteriorating and that death was expected, although it was also consistent with digoxin toxicity.

(14) RICHARD McKEIL

Diagnosis

This six week old baby boy suffered from a transposition of the main arteries of the heart, but in addition, both arteries came off the right ventricle. There was also a problem with the mitral valve (the valve connecting the left atrium and ventricle), in that it tended, when open, to send blood to the right ventricle. This condition is known as "straddling the mitral valve." He also suffered from a patent ductus arteriosus, coarctation (narrowing) of the aorta, and a ventricular septal defect.

Prognosis

The views of the cardiologists were as follows: Dr. Rowe - high risk death, Dr. Nadas - guarded, Dr. Hastreiter - 6 (out of 10).

Course of Treatment

This infant was admitted to the Hospital on September 2, 1980. An operation was performed on September 22 to divert the blood from the right pulmonary veins into the right atrium, and to band the pulmonary artery to reduce the amount of blood flowing back to the lungs. The infant tolerated the operation well; the surgeon gave him a good prognosis, and he was returned to the ward from the I.C.U. the next day. His early post-operative course appeared satisfactory but, when back on the ward, he continued in congestive heart failure, fed poorly, and vomited persistently. He developed an infection and on September 29 was transferred to the Infectious Diseases Ward (7C/D). He returned to Ward 4A on October 6. He was still suffering from congestive heart failure, feeding poorly, and vomiting. A second catheterization was scheduled for October 16.

Terminal Events

On October 14, his vital signs were normal but he vomited twice and was placed on intravenous fluids. Later that night he appeared alert and bright, although somewhat pale. At 3:45 a.m. October 15, his cardiac monitor went off indicating a drop in the heart rate. A doctor was called, but the heart rate sank again, and the baby went into cardiac arrest from which he could not be resuscitated. The terminal events included extreme bradycardia, gasping, cyanosis, and ventricular fibrillation. He was pronounced dead at 4:27 a.m.

Autopsy

The autopsy confirmed the clinical diagnosis and found the shunt and the banding intact. Death, in the heading of the Final Autopsy Report, was attributed to: "congenital heart disease: post-operative cardiac failure".

Toxicology

Digoxin was used regularly to try to control his cardiac failure, but the baby had some difficulty tolerating it. It must also be remembered that this baby was accidentally given his proper dose, but after a "hold" order on September 10. A level of 4.6 ng/ml was reported on September 16, but on September 24 it had fallen to 2.5 ng/ml. In early October his readings were 1.9, 3.4, 1.2 and 1.3 ng/ml. A sample taken on October 14, the day before his death, resulted in a level of 5.6 ng/ml. It is possible that this sample was taken too soon after dosage to give a true reading. Similarly, a problem of sample timing has been suggested as the explanation for the earlier level of 3.4 ng/ml. In any event, no digoxin was to have been given to him after the dose at 6:00 a.m. on October 14. No post-mortem tests were performed.

The Experts

There was no doubt that this was a sick baby, but on the other hand, there was every intention of discharging him once his feeding problem improved. The Hospital doctors attributed death to his clinical condition, but all agreed that digoxin toxicity may have contributed. Dr. Bain and Dr. Rowe considered the death somewhat surprising since the infant seemed reasonably well on the evening prior to his death. Dr. Rowe also stated that the ventricular fibrillation which occurred during the terminal events might have been caused by the myocardial necrosis seen on autopsy. Dr. Hastreiter thought there was a fair probability of digoxin overdose and Dr. Fay thought it was a suspicious death. Dr. Nadas, in his report to Atlanta, found the baby's death unexpected but consistent with both his clinical condition and digoxin toxicity.

(15) ANTONIO ADAMO

Diagnosis

This nine day old baby boy had a condition known as "dextrocardia" wherein the heart was displaced towards the right side of the body. This reversal was not important for his cardiac condition, but he also suffered from a transposition of the great arteries, with both coming off the right ventricle, and a large ventricular septal defect. There was also a narrowing of the pulmonary artery. Because of these conditions, there was a mixing of blue and oxygenated blood going through the aorta to the body. This child was getting some oxygenated blood, but not enough. He was also noted to have mild dysmorphic physical characteristics.

Prognosis

The views of the cardiologists were as follows: Dr. Rowe - high risk death, Dr. Nadas - guarded, Dr. Hastreiter - 7 (out of 10).

Course of Treatment

The child was admitted to the Hospital on October 14, 1980, at four days of age. He was then experiencing the beginnings of cyanosis; he was intermittently dusky and pale in colour. He underwent catheterization on October 15, and despite his very tender age, surgery (Blalock-Taussig shunt) was performed the next day. The baby tolerated the operation and was back on the ward within forty-eight hours. There, however, he experienced periods of rapid breathing and fast heart rate; digoxin and diuretics were administered with full digitalization completed the morning of October 19. A proposed increase in the digoxin dosage was scheduled for the evening of October 20.

Terminal Events

On October 19, during the day he fed poorly, his duskiness continued, and his breathing was laboured. At 4:15 p.m., a naso-gastric tube was passed for feeding. Seizure-like activity, ventricular fibrillation, junctional rhythm (an abnormal heart rhythm which does not originate in the sinus node), and bradycardia occurred. Resuscitation efforts were initially partly successful, but at 5:43 p.m. he was pronounced dead.

Autopsy

Parental consent was not given and no autopsy was performed.

Toxicology

While the baby was on digoxin for his condition, no ante-mortem or post-mortem levels were taken. A level was ordered for October 20, but the baby did not survive long enough for the test to occur.

The Experts

The Hospital doctors agreed that the terminal events were consistent both with the baby's clinical condition and with digoxin toxicity. Dr. Izukawa thought the cardiac arrest may have been precipitated by the insertion of the naso-gastric tube, as did Dr. Mirkin and Dr. Freedom. Dr. Mirkin also considered the death to be unexpected. Dr. Rowe testified that the child had been deteriorating over a period of 24-48 hours prior to his death. He would have expected the baby to do quite well following his surgery and was a bit surprised that he went into heart failure post-operatively. Overall, he felt the baby's death was attributable to his congestive heart failure. Dr. Fay considered digoxin toxicity to be a low suspicion, while Dr. Hastreiter thought it to be a fair probability. Dr. Nadas, in his report to Atlanta, considered this death unexpected but consistent with the baby's clinical condition. He expressed special concern regarding digoxin intoxication.

(16) FRANCIS VOLK

Diagnosis

This baby boy spent the whole of his three month life in hospital. He suffered from hypoplastic (underdeveloped) right lung and right pulmonary artery, dextral position of the heart, an atrial septal defect, coarctation of the aorta, and patent ductus arteriosus. He also showed some dysmorphic features. In short, he had not only pulmonary problems but heart problems as well, and some of the latter were the result of the small right lung causing an overextension of the left lung which pushed the heart over to the right side. He was also believed to suffer from partial DiGeorge syndrome which indicates a lack of part of the thymus gland resulting in a deficiency in the baby's immune system.

Prognosis

The views of the cardiologists were as follows: Dr. Rowe - high risk death, Dr. Nadas - poor, Dr. Hastreiter - 8 (out of 10).

Course of Treatment

The baby underwent at least two operations, one at one month to repair the aorta (coarctectomy) and the ductus, and one at ten weeks to remove the right lung (pneumonectomy.) After the second operation, the baby suffered two serious cardiac arrests on October 12, 1980, perhaps brought on by his respiratory problems. In each case he was successfully resuscitated and he was returned to the ward on October 20. Two days later he was experiencing laboured and irregular breathing, was very pale and restless. He was being fed by naso-gastric tube and was placed on a cardiac monitor.

Terminal Events

On the day of his death, October 23, his respirations became more laboured and at 2:00 p.m. he vomited and required suctioning. He seemed to recover but then deteriorated rapidly. A final cardiac arrest from which he could not be resuscitated took place at 4:50 p.m.

Death was pronounced at 5:15 p.m. The terminal events were marked by seizures and ventricular fibrillation.

Autopsy

A partial autopsy conducted on the child's heart and lungs attributed death to acute bronchopneumonia and congestive heart failure.

Toxicology

The baby was on digoxin. He had experienced high levels in August and September, but the last test on September 22 was in the therapeutic range. No further tests were taken in the month prior to death. The only post-mortem test was on fresh-frozen skin tissue, with a level reported to be in the therapeutic range.

The Experts

All of the experts agreed that the baby was very sick and died of natural causes. Dr. Rowe stated that in this case the above-mentioned ventricular fibrillation was part of the normal result of the resuscitation effort itself; it was not a manifestation of digoxin toxicity. Dr. Nadas, in his report to Atlanta, rated the infant's death unexpected but consistent with his clinical condition. As noted in Chapter 4, the joint conference of surgeons and cardiologists of January 12, 1981, also classified the death as "unexpected", i.e., not inevitable. However, the terminal events were not considered to be consistent with digoxin toxicity by Dr. Nadas or others. The Atlanta Report placed the baby's death in Category C, the natural death category.

(17) MATTHEW LUTES

Diagnosis

This twenty-eight day old baby boy suffered from coarctation (narrowing) of the aorta, right ventricular hypertrophy, two ventricular septal defects, a defect in the atrial septum, cardiomegaly (enlarged heart), failure to thrive, and had dysmorphic features. He was admitted to the Hospital five days before his death with congestive heart failure, the result of his cardiac defects.

Prognosis

The views of the cardiologists were as follows: Dr. Rowe - high risk death, Dr. Nadas - guarded, Dr. Hastreiter - 6 (out of 10).

Course of Treatment

On admission on November 12, 1980, the child had fast shallow respirations and was in mild congestive heart failure. An echocardiogram and a cardiac catheterization performed on the same day confirmed the ventricular septal defects noted above and disclosed evidence of moderate pulmonary hypertension (high blood pressure in the pulmonary circulation). Surgery was contemplated but withheld pending the results of chromosomal studies and improved nutrition (it eventually proved that he did have chromosomal abnormalities); an X-ray showed the existence of pulmonary edema (fluid in the lung). Attempts were made to control the congestive heart failure with diuretics and the use of digoxin, but it was apparent by November 15 that the treatment was not entirely successful - the baby had an enlarged liver, respiratory distress, and was vomiting, although he showed no signs of fever, and his vital signs were stable. By November 16 he was restless with an increasing respiratory rate. Nevertheless, that night he appeared better, tolerating feedings and settling well.

Terminal Events

At midnight, November 16, the baby became diaphoretic (perspiring) and pale and was again vomiting. His heart rate increased and his respirations became more shallow.

By 12:30 a.m. he became severely bradycardic. At 12:50 a.m. he suffered cardiac arrest from which he could not be resuscitated. Death was pronounced at 1:34 a.m.

Autopsy

The autopsy confirmed the various cardiac defects. The Final Autopsy Report made no specific finding as to cause of death and is headed simply: "congenital heart disease".

Toxicology

There had been some concern that even a digoxin level of 2.1 ng/ml (measured on November 14) was too high for this baby and that his vomiting might have been caused by an adverse reaction to therapeutic doses of digoxin. In consequence, the dosage of digoxin was reduced on November 14 and a "hold" order was made on November 15, but the drug was restarted on November 16 since the baby's cardiac problem required its use. Post-mortem digoxin levels taken on fixed heart and lung tissues and tested by RIA plus HPLC and RIA were considered inconclusive by Mr. Cimbura although the lung tissue reading was in the overlap area: potentially either therapeutic or toxic.

The Experts

The sudden onset of the terminal events and the bradycardia could have been indicative of digoxin toxicity but were also perfectly consistent with his clinical condition. Dr. Fowler described his cardiac condition as "lethal". Dr. Nadas, in his report to Atlanta, rated the infant's death unexpected but consistent with both his clinical condition and digoxin toxicity. Dr. Rowe indicated that he was absolutely satisfied that the course of the child's death resulted from his cardiac malformations. Dr. Fowler agreed. Dr. Fay maintained some suspicion of digoxin toxicity; Dr. Hastreiter thought the possibility almost nil.

(18) JOHN ONOFRE

Diagnosis

This eighteen day old baby boy had the most severe type of tetralogy of Fallot including a large ventricular septal defect. There was no exit to the pulmonary artery from the right ventricle, the only entrance to the artery being via the ductus arteriosus, and there was a narrowing of the pulmonary artery. The result was that very little blood got to the lungs, and what little there was would cease with the closing of the ductus. In essence his was a classic "blue baby" condition.

Prognosis

The views of the cardiologists were as follows: Dr. Rowe - high risk death, Dr. Nadas - guarded, Dr. Hastreiter - 5 (out of 10).

Course of Treatment

The child was admitted to the Hospital on November 22, 1980, at one day of age because of irregularities in his heart rate, cyanosis and suspected congenital heart deformity. On the following day, a cardiac catheterization procedure led to the diagnosis of tetralogy of Fallot. On November 24 a Blalock-Taussig shunt operation was performed. The surgeon reported that: "He underwent this procedure without problem and was sent to the I.C.U. in good hemodynamic status". He had a relatively uneventful post-operative course although a bloody stool and possible gastrointestinal infection were noted on his fourteenth day. He suffered from arrhythmias throughout his hospital stay. He was initially treated with digoxin but this was discontinued on December 4 as it did not appear to be countering the arrhythmias. His condition was relatively stable on December 7 and 8 although he was to receive nothing by mouth.

Terminal Events

On December 9, at 3:20 a.m., he suddenly developed bradycardia and arrhythmia, in particular junctional rhythm, progressing to cardiac arrest. He was pronounced dead at 4:10 a.m.

(JOHN ONOFRE)

Autopsy

The autopsy disclosed that the shunt was intact and there was evidence of various other precipitating causes of death including sepsis (infection), necrosis of part of the heart (death of tissue), and myocardial (heart muscle) degeneration. The pathologist thought death from electrical instability or Escherichia coli (usually known as E. coli) septicemia (particular bacterial toxins in the blood) was possible and may have contributed to it significantly. He described the death as "somewhat sudden and unexpected" in the Final Autopsy Report. It was reported to the Coroner, but not until the summer of 1981 by which time of course the investigation was well underway.

Toxicology

The baby's last digoxin reading on December 2 was well within the therapeutic range at 1.1 ng/ml. The body was exhumed for tissue testing almost two years later and liver and tongue tissues were found to contain digoxin as was a thigh muscle specimen, all tested by RIA plus HPLC and RIA. Besides the inherent unreliability of levels measured in exhumed tissues, this body had been embalmed, and after such a long burial the tissues had decomposed rendering the digoxin test results inconclusive. In any event, Dr. Kauffman considered that none of the levels measured was inconsistent with the digoxin received by the infant prior to its discontinuance.

The Experts

There was no unanimity as to the cause of death. It was a sudden death without immediate cause which prompted Dr. Nadas, in his report to Atlanta, to classify the death as unexpected and inconsistent with the baby's clinical condition. He also rated the death consistent with special concern regarding digoxin toxicity. Dr. Fay considered the death suspicious; Dr. Freedom would accept the autopsy findings as well as the possibility that the shunt was too small to sustain life. Dr. Rowe said that the baby's condition was not one which would make death inevitable, and he and Dr. Bain said that his death was unexpected at the time it in fact occurred. With the autopsy findings in hand however, they felt the child's death had been adequately explained by his underlying clinical condition. Dr. Kauffman noted there was no objective pharmacologic evidence to support the theory that this infant died from digoxin intoxication. Dr. Hastreiter opined that neither the sepsis nor the chronic arrhythmias would have caused the baby's death. He considered the case one of probable digoxin intoxication.

(19) DARCY MacDONALD

Diagnosis

This five month old baby boy suffered from Down's syndrome (mongolism) a condition producing mental retardation. He was admitted to the Hospital on December 12, 1980, only fifteen or sixteen hours prior to death. He suffered from breathing difficulties, but he also had both atrial and ventricular septal defects resulting in an enlarged pulmonary artery, the mixing of red and blue blood, and excessive circulation of blood to the lungs.

Prognosis

The views of the cardiologists were as follows: Dr. Rowe - high risk death, Dr. Nadas - guarded, Dr. Hastreiter - 5 (out of 10).

Course of Treatment

The child had been treated at the referring hospital for congestive heart failure and respiratory infection. Digoxin, ampicillin, and diuretics were prescribed and administered. On transfer to the Hospital, maintenance doses of digoxin were prescribed and one dose was given. Pneumonia was found; his chest was noisy and congested, his colour poor, and he vomited his evening feeding. Because of his short stay at the Hospital there was no time to attempt any cardiac repair, but the Hospital would in the ordinary course have expected to undertake it with reasonable hope of success. However, at midnight after admission, it was noted that his heart rate was variable. The baby was placed on a cardiac monitor. The baby experienced one episode of slow heart rate and a doctor was notified; the infant responded to stimulation and then slept comfortably.

Terminal Events

At 3:35 a.m. December 13, he was still pale and started to cough and spit up mucous. He was suctioned, but immediately went into cardiac arrest from which he could not be resuscitated. Death was pronounced at 4:30 a.m.

(DARCY MacDONALD)

Autopsy

The Preliminary Autopsy Report gave as the principal finding in the heading: "congenital heart disease". The Final Autopsy Report eliminated this and substituted as the main findings in the heading: "Down's syndrome - viral pneumonia". No immediate cause of death was stated in either report. The Hospital's Discharge Report set out no definite cause of death but suggested a vagal reflex caused by suctioning, arrhythmias, or poor sinus function as possible causes.

Toxicology

As noted above, the baby had been on digoxin previously and received one dose at the Hospital for Sick Children. There were no ante-mortem or post-mortem digoxin tests performed.

The Experts

All the experts, except possibly Dr. Mirkin, agreed that digoxin toxicity was possible as a cause of death. Indeed the resident attendant at death had named it as one of the possible causes of death. The possibility of digoxin toxicity arising from prescribed doses administered at the referring hospital was not supported by the electrocardiogram results obtained on admission to the Hospital. Drs. Rowe, Fowler, and Bain all thought the death fully explained by the child's clinical condition including the pneumonia. Dr. Nadas, in his report to Atlanta, rated the boy's death unexpected, although consistent with his clinical condition, and also with special concern regarding digoxin toxicity. Dr. Hastreiter did not believe that the pneumonia was severe enough to cause death in this case, although it may have contributed. He thought digoxin toxicity probable. Dr. Fay considered it but thought natural death more likely. Dr. Mirkin thought digoxin intoxication very unlikely. All the medical experts agreed the death was unexpected.

(20) REAL GOSSELIN

Diagnosis

This three week old baby boy was transferred on December 17, 1980, the day before his death, from a hospital in Winnipeg where he had been diagnosed after cardiac catheterization as having congestive heart failure and an interrupted aortic arch. At the Hospital for Sick Children, it was discovered that the aorta was not totally interrupted but was underdeveloped and severely narrowed. Such an obstruction can possibly be tolerated so long as the ductus is patent, because there is room there for passage through a bypass, but once the ductus closes the dimension of the aorta is reduced further. The condition is potentially operable but, without surgical intervention, death is inevitable. In this baby's case it was apparent that the ductus was patent and had become the major supplier of blood to the lower body. There were other less severe defects such as mitral and aortic valve abnormalities. In addition, his left ventricle was undersized.

Prognosis

The views of the cardiologists were as follows: Dr. Rowe - high risk death, Dr. Nadas - guarded, Dr. Hastreiter - 8 (out of 10).

Course of Treatment

The child had been treated with digoxin and furosemide in Winnipeg. Prostaglandin therapy was started at the Hospital to keep the ductus open but surgery was clearly necessary. There is some confusion about the timing of the proposed operation. The parents of the baby understood it was to be performed on the morning of December 17. The doctors at the Hospital maintain that it was not to be performed until December 18, because on admission the baby had a high digoxin level (3.7 ng/ml). In any event it was scheduled for December 18. The parents claim that they were also told by doctors at the Hospital not to be concerned about the delay because the child was stable, and if he deteriorated, there would be plenty of time to get him to the Operating Room.

(REAL GOSSELIN)

Terminal Events

There was not time for an operation. At approximately 7:00 p.m. on December 17 the baby experienced two episodes of apnea, accompanied by bradycardia and lethargy. He was monitored very closely and given furosemide. Thereafter, his condition was relatively stable although he continued to have periods of irregular breathing and bradycardia. As it happens, the parents were present with the baby when his monitor went off at about 2:00 or 2:15 a.m. on December 18. The baby was having a serious bout of bradycardia and irregular heart rate, accompanied by very shallow respirations. A doctor was summoned. The baby continued to be bradycardic, and a Code 25 was called. In spite of efforts at resuscitation, no electrical response could be achieved despite stimulation and he was pronounced dead at 3:16 a.m.

Autopsy

The autopsy disclosed the major aortic problem and other relatively minor heart defects and also noted that the ductus was patent. The Final Autopsy Report is headed "congenital heart disease: coarctation of aorta", but no specific cause of death is given in the body of the text.

Toxicology

As noted above, a digoxin level taken on arrival at the Hospital for Sick Children was 3.7 ng/ml. This brought about an immediate "hold" order. No digoxin was thereafter administered. No further levels, ante-mortem or post-mortem, were taken. Although the doses administered in Winnipeg were felt by the doctors at the Hospital for Sick Children to be on the "high-side", no one expressed the view that they were sufficiently high to produce extreme toxic symptoms; indeed, Dr. Rowe's opinion was to the contrary.

The Experts

Dr. Freedom wrote a letter to the referring physician on the day of the child's death in which he said:

> . . . I am really disturbed by this baby's demise just a few hours prior to surgery. I doubt that the demise can be explained purely on the basis of apnea secondary to the prostaglandin therapy, and at this time, I really don't have a good explanation for this baby's sudden deterioration and death.

In testimony, however, Dr. Freedom explained that remark by saying that at the time he wrote the letter he was under the misapprehension that the baby's response to the prostaglandin therapy had been good. Indeed, Dr. Stephen who wrote the Discharge Report as much as said so. Later, on Dr. Freedom's own review of the child's chart, he concluded that the opposite had been the case. The blood pressure in the lower extremities remained consistently lower than that in the upper indicating that blood was not flowing freely past the obstruction. He would rule out digoxin overdose, but all the outside experts found the death consistent with digoxin toxicity. Dr. Nadas, in his report to Atlanta, rated the infant's death unexpected, inconsistent with his condition, and consistent with digoxin toxicity, but the other experts generally were of the opinion that the death, while unexpected as to time, was consistent with the baby's clinical condition. Dr. Hastreiter thought there was a good probability of digoxin toxicity. He conceded that the failure of the prostaglandin therapy (if it had indeed occurred) could have precipitated death. Dr. Fay originally thought digoxin toxicity the cause, but he based it largely on Dr. Freedom's letter, and changed his mind when he heard of Dr. Freedom's change of opinion. Dr. Mirkin considered the baby not to be at risk at the time and the death unexpected.

(21) STEPHANIE LOMBARDO

Diagnosis

This ten day old baby girl suffered from tetralogy of Fallot including a large ventricular septal defect and stenosis of the pulmonary artery. She also had an extremely small main pulmonary artery and branches. The result was that not enough blood was getting into the lungs, and corrective surgery (a Blalock-Taussig shunt) was proposed.

Prognosis

The views of the cardiologists were as follows: Dr. Rowe - high risk death, Dr. Nadas - guarded, Dr. Hastreiter - 6 (out of 10).

Course of Treatment

This baby was admitted to the Hospital on December 13, 1980, the day of her birth, when she was noted to be cyanotic and a congenital heart defect was suspected. She was initially admitted to the neonatal ward (for infants less than thirty days of age) and underwent a cardiac catheterization two days later. At four days of age, on December 17, she underwent an operation to place a shunt between the aorta and pulmonary arteries, and it was a somewhat unusual shunt made necessary by the condition of her arteries. She tolerated the operation well, spent five days in the I.C.U., and was returned to the ward on December 22. There was some concern about the size of the shunt and she was treated with the drug heparin to prevent its occluding (closing). There was also some concern about the oxygenation of her blood but that concern lessened, and the supplementary oxygen she was receiving was discontinued that morning. She remained slightly cyanotic, except when resting when her colour improved, and she was otherwise stable and feeding quite well. Digoxin was never prescribed at the Hospital, and some experts suggested it would in fact have been contra-indicated for this child.

Terminal Events

Throughout the late evening on December 22 the baby remained relatively stable; her apex was regular and normal, her colour remained satisfactory although she was dusky when upset, she fed eagerly and settled well. She continued to receive heparin for her shunt through an intravenous and sage pump apparatus (a pump which regulates the flow of medication). She was not receiving any other medication of any kind. At about 3:30 a.m. on December 23, she was noted to be suddenly bradycardic, and to have an irregular heart rate and shallow breathing. She was placed immediately on a cardiac monitor. She became increasingly dusky in colour and her extremities were cyanotic. Minutes later she vomited, and at about 3:45 a.m., that is about twelve hours after returning to the ward, she suffered a cardiac arrest and was pronounced dead at 4:20 a.m.

Autopsy

Permission for an autopsy was refused and none was performed. As will be seen, however, the baby was exhumed in February, 1982, in the middle of the Preliminary Inquiry into the charges against Susan Nelles.

Toxicology

As noted above, the baby was never on digoxin. The exhumation and subsequent testing at the Centre of Forensic Sciences, some fourteen months after death, revealed substantial amounts (indeed the highest levels found by Mr. Cimbura in any exhumed tissue) of digoxin in her chest fluid, heart, liver, lung, muscle, stomach, and bowel tissues, all tested by RIA plus HPLC and RIA. In addition, the child's chest fluid and heart tissue specimens were tested by GC/MS. As I have indicated before, the amount is not so important; the significant thing is the presence of the drug when none was prescribed or known to have been administered.

Medication Error

As I have said, there is always a possibility of an individual mistake. Here, however, it must be appreciated that there could not have been error in the amount of the digoxin dose administered because the child was not on the drug. Dr. Spielberg from the Hospital, however, testified that a medication error may have been made shortly before or during the resuscitation effort. Alternatively, it was suggested by Dr. MacLeod that one dose of digoxin administered in error to the child at any time during her ten day hospitalization could account for the presence of digoxin later found in her exhumed tissues. In the opinion of Dr. Hastreiter, however, a maintenance or therapeutic dose of digoxin (less than one-tenth of one adult ampoule) given in error could not account for the levels found in the child. Certainly there was no indication of digoxin toxicity until her terminal events. Dr. Kauffman agreed with Dr. Hastreiter and expressed the opinion that a medication error was unlikely. It is important to note that heparin was the only drug being administered to the baby in the days immediately prior to her death. Heparin, although available on the wards in ampoules similar to those used for the intravenous preparations of digoxin, was given in such minute quantitites that, had a similar amount of intravenous digoxin been given, it would have had no appreciable effect. The child was receiving no oral medications so that it is difficult to contemplate a mistake involving oral administration. It would seem that any error would necessarily have involved a mistake in identity. It is also important to note as well that this child died when there were fewer patients and staff on duty because of the holiday season and there was, therefore, less likelihood of confusion and consequent medication error.

The Experts

Dr. Rowe described this child's final course as an "immediate, rapid and dramatic decline" which, at the time it occurred, was sudden and unexpected. When she died, the Hospital doctors put the cause of death down to shunt occlusion. A day before the baby's transfer to the ward, Dr. Izukawa noted in the chart his concern about her post-operative course in the I.C.U. Because there was no autopsy, and because of the deterioration of the body in the long period between burial and exhumation, the theory of shunt occlusion could be neither proved nor disproved. Certainly all the experts agreed that the terminal events were consistent therewith, as they were with digoxin toxicity. Dr. Nadas, in his report to Atlanta, rated the infant's death unexpected, consistent with her clinical condition, and consistent with digoxin toxicity. The presence of the unprescribed digoxin in the tissues, however, persuaded the Hospital doctors and Dr. Mirkin that digoxin toxicity was a possible cause of death, and Drs. Hastreiter and Kauffman said that it was the probable cause.

(22) JESSE BELANGER

Diagnosis

This forty-one day old boy was afflicted with pulmonary stenosis and what was essentially a single ventricular chamber and a single atrial chamber with only one valve between them. It followed that there was a mixing of blood in each chamber. The pulmonary artery was much smaller than the aorta, and the latter led off to the right side of the body. In addition he had several non-cardiac defects including a cleft lip and palate, a deformed ear, a partial DiGeorge syndrome (deficiency in the body's immune system), arhinencephaly (absence of a portion of the brain), and a deviation of the eyes which might well have led to blindness.

Prognosis

The views of the cardiologists were as follows: Dr. Rowe - high risk death, Dr. Nadas - guarded, Dr. Hastreiter - 8 (out of 10).

Course of Treatment

Because more blood from the common ventricle was flowing through the aorta, the body was not getting the oxygenated blood it needed. The baby was admitted to the Hospital on November 19, 1980. His position was not precarious and surgery was delayed for a month pending chromosomal studies. A Blalock-Taussig shunt was inserted surgically on December 22 on the left side between the aorta and the pulmonary artery. The operation appeared to improve the oxygenation and post-operatively the baby was in stable condition, although there was fear concerning the adequacy of the shunt. Heparin was prescribed to keep the shunt open. On December 26, the baby was moved from the I.C.U. to Ward 7G because of a bed shortage in the I.C.U., and two days later he was transferred to Ward 4A/B. At the time, Wards 4A and 4B were joined during the holiday period and only one nursing team was on duty.

Terminal Events

The baby appeared stable on the ward during the early afternoon of December 28 with good colour and a regular apex; he tolerated a naso-gastric tube feeding shortly after arrival on the ward. Five and one-half hours after admission to Ward 4A/B (this was one of the few babies who did not die in the early morning), during a second feeding, he suffered bradycardia and successive arrhythmias; his colour became extremely poor and his respirations shallow, leading to a cardiac arrest from which he could not be resuscitated. He was pronounced dead at 8:16 p.m.

Autopsy

The autopsy confirmed the diagnosis of cardiac and other defects and found the Blalock-Taussig shunt "intact and patent". No specific cause of death was given. The Final Autopsy Report listed as the main finding: "congenital heart disease".

Toxicology

The baby was not on digoxin so no ante-mortem levels were taken. Eighteen months after death (four weeks after the discharge of Susan Nelles), the body was exhumed and tests were made on his muscle tissue using RIA plus HPLC and RIA, and on his liver tissue using GC/MS as well as RIA and HPLC and RIA. The tests were positive for digoxin in each case. The muscle reading was within the range of therapeutic concentrations reported in fresh autopsy specimens of children on digoxin therapy, but the liver level was well above that range. For the reasons I have outlined, the concentrations were not taken by Mr. Cimbura as anything more than evidence of the presence of unprescribed digoxin in the body.

Medication Error

As in the case of Stephanie Lombardo, Dr. Spielberg suggested that the baby might have received a dose of digoxin in error before or during the resuscitation effort. Dr. MacLeod expressed the view that the accidental dose might have been administered at any time during the child's thirty-five day hospitalization. Dr. Hastreiter, however, considered that the levels measured meant that neither one maintenance dose nor even one loading dose could account for the levels of the drug found. Dr. Kauffman essentially shared the same view. He regarded the possibility of medication error unlikely as did Dr. Mirkin. Repeated medication error was clearly most unlikely. It is important to note that there were no symptoms of digoxin toxicity before the onset of the baby's terminal events.

The Experts

All the experts, except Dr. Freedom, conceded that the death was consistent with digoxin toxicity, but all but Dr. Nadas found it also consistent with his clinical condition. Dr. Nadas, in his report to Atlanta, rated the baby's death unexpected, inconsistent with his clinical condition, and consistent with digoxin toxicity. In view of the unprescribed digoxin, most of the experts, including all from outside the Hospital, classified the death as induced by digoxin toxicity. Dr. Kauffman thought there was a high probability that digoxin contributed directly to the baby's death as did Dr. Hastreiter. Dr. Mirkin described it as possible.

(23) JANICE ESTRELLA

Diagnosis

This four month old baby girl was afflicted with Down's syndrome as well as with a complete atrioventricular defect (absence of the septum between the left and right sides of the heart). In addition, instead of two valves between the atria and the ventricles, there was only one. There was also a patent ductus arteriosus. The result of this in the circulation was that blood came to the right atrium in the ordinary way but was immediately mixed with the blood from the lungs in the left atrium. In consequence, too much blood was pumped out to the lungs, enlarging the pulmonary artery, and the blood passing into the aorta was not completely oxygenated.

Prognosis

The views of the cardiologists were as follows: Dr. Rowe - high risk death, Dr. Nadas - guarded, Dr. Hastreiter - 8 (out of 10).

Course of Treatment

When the child was first in the Hospital she was considered too young for surgery but was readmitted on December 14, 1980 - at thirteen weeks of age - and repair effected of her ductus and septal defect two days later. She had a stormy post-operative course with some collapse of the right lung. On December 28, she was returned to the ward in relatively stable condition, although she was not feeding well and needed a naso-gastric tube. There was some evidence from time to time of heart failure (including an incident on January 7, 1981, leading to an arrest from which she was resuscitated - discussed infra under "Toxicology") and, as well, symptoms of nutritional problems and possible pneumonitis for which she was administered antibiotics. She was receiving digoxin until January 7 when it was ordered held - see discussion infra - and diuretics throughout. Earlier indications of possible renal impairment were countered with administration of furosemide. By January 10, however, she was considerably improved and there was no suggestion of heart rate irregularity or congestive heart failure according to the resident physician who cared for her. Although she was still seriously ill and on constant care nursing, her apex and other vital signs were normal on the night shift of that day. There was some temperature problem, and some tachypnea, but she was sleeping and tolerating tube feedings well. Notwithstanding the resident's recorded opinion, Dr. Rowe stated that the infant's cardiologist considered her to be still in a condition of severe heart failure.

Terminal Events

At 2:40 a.m. on January 11, the baby was observed to be gasping, with a rapidly declining respiratory rate and bradycardia. Little heart beat could be heard. The cardiac arrest team was called but the baby could not be resuscitated. She was pronounced dead at 3:22 a.m.

Autopsy

There is a note in the Hospital records indicating that the death was reported to the Coroner but there is no record of this in the Coroner's office, and the autopsy was not conducted under his supervision. It was conducted by Dr. Taylor under the supervision of Dr. Mancer. Usually an interim report is prepared immediately afterwards, and a final report (which may not differ greatly), after all tests have been completed. In this case only the Final Autopsy Report is found in the child's chart. It does not list a principal finding in its heading but does relate the course of her treatment and surgery, notes her cardiac and extracardiac defects, manifestations of marked congestive heart failure, and evidence of acute bronchopneumonia, and concludes in its final two paragraphs as follows:

> The surgical repair of the congenital heart defect appeared intact at autopsy examination. A small perforation of one mitral leaflet was present. This may have contributed slightly to the congestive heart failure through some degree of mitral regurgitation. No specific explanation for the congestive heart failure other than

> the presence of a severe congenital heart anomaly (though repaired) was found.
>
> Samples of post mortem blood were obtained for assay of digoxin levels. These samples were contaminated slightly by edema fluid and ascitic fluid. The digoxin levels on these samples measures [sic] 72.0 ng/ml (toxic range 2.0 to 9.0 ng/ml blood). This level is markedly elevated over the normal therapeutic range, and if accurate would explain the death of the patient.

Toxicology

This baby was, of course, on digoxin for most of her life and regularly monitored for digoxin levels. On January 7, the baby suffered an attack of bradycardia. The nurses and doctors attending feared an imminent cardiac arrest and called the arrest team. The baby was successfully resuscitated. Digoxin toxicity was suspected and a blood sample taken; the level was found to be greater than 5.0. I point out here that 5.0 ng/ml - sometimes 4.7 ng/ml - was the highest level for which the Hospital RIA equipment was calibrated. To find the actual level, it was necessary to dilute the sample one or more times until a reading was obtained within the scale of the equipment. The proper level then became that reading multiplied by the dilution factor. It developed that with this sample only one dilution was made and the reading was still beyond the scale of the equipment, i.e., it was greater than 9.4 ng/ml. No further dilution could be made because none of the sample remained available for testing. Digoxin was of course held and never again restarted. A level taken on January 8 was 7.8 ng/ml and on January 9, 4.7 ng/ml. No further levels were taken prior to her death.

After the death, as I have said before, a post-mortem test - perhaps the first in the Hospital's history - was performed under the following circumstances. Dr. Taylor testified that he was instructed by Dr. Freedom to take a post-mortem sample for digoxin testing but that Dr. Freedom did not fully explain why and certainly did not indicate any fear of digoxin toxicity. As Dr. Taylor remembered it, Dr. Freedom simply indicated that there had been some difficulty in controlling the child's digoxin levels during life and the post-mortem specimen was to be obtained for a cross-check. Dr. Freedom had no recollection of the instruction, but it would have been a reasonable thing for him to do in light of the child's recent high digoxin levels, and I cannot imagine Dr. Taylor inventing it. In any event, Dr. Taylor forgot about his instruction until after the autopsy which commenced about 2:50 p.m. and was completed between 6:00 and 7:00 p.m. on January 11. He then, in company with a colleague, reattended at the morgue and took blood samples from the leg vein and from the pelvic cavity. If he had taken the sample in the course of the autopsy, he would have drawn it from the superior vena cava, but by the time he got around to it, all the organs had been removed and the blood vessels had been cut, and he was afraid he could not get a suitable specimen from the usual source. He therefore went to the leg vein; he took precautions to prevent contamination including cleaning the site and thought that the sample obtained was free from any significant contamination. He doubted, however, that he had obtained sufficient blood for testing and therefore took a further sample from the pelvic cavity.

Dr. Taylor kept the two samples separate and took them to the Biochemistry Laboratory for testing. Although he did not realize it at the time, the samples remained apart and were tested separately. The final results came back as greater than 4.7 ng/ml for the leg vein and (after many assays) 72 ng/ml for the pelvic cavity. He assumed the 72 ng/ml reading was the diluted determination of the earlier "greater than 4.7 ng/ml." In any event he had never heard of a level so high. He discussed it with his colleagues, and later with Dr. Freedom, who agreed with him that the high level was probably caused by an error either mathematical or induced by an artefact, for example, contamination. Dr. Freedom may have suggested to Dr. Taylor that he (Dr. Taylor) check out the reading with the biochemist but this was not followed up. Dr. Taylor did, however, report the reading to Dr. Mancer who wrote into the Final Autopsy Report the last paragraph quoted above. As earlier set out, the contamination of the pelvic cavity sample was described by Drs. Mancer and Taylor in the Final Autopsy Report as "slight". No differentiation was made between the leg vein and the pelvic cavity samples. According to Dr. Rowe none of the other cardiologists at the Hospital learned of the post-mortem level of 72 ng/ml until the Final Autopsy Report was sent to Dr. Fowler in approximately the second week of March, 1981.

Both Dr. Kauffman and Dr. Spielberg estimated that on the basis of the readings taken after the January 7 incident, and the fact that digoxin was withheld thereafter, the level in her blood (failing an unprescribed dosage) would have been less than 3 ng/ml at death.

Some examination of fixed heart tissue specimens was made at the Centre of Forensic Sciences using RIA and HPLC and RIA but the results (indicating only small amounts of digoxin), like so many of the tissue tests, were inconclusive. The tissue specimens had been fixed in preservative solution for several months.

The Experts

All Hospital doctors assumed the death to be natural at the time. After the police investigation started in March, almost all the experts were satisfied that the death was the result of digoxin overdose. While the death was unexpected as to time, the terminal events were consistent with both the anatomical condition of the baby and digoxin toxicity. The reading of 72 ng/ml was, however, the deciding factor. The validity of that reading was accepted after the accuracy of the tests had been confirmed by the biochemists.

At the Preliminary Inquiry the experts were satisfied with the readings and indeed Dr. Taylor suggested that the effect of any contamination might be to reduce the digoxin level as a result of the dilution of the blood by other fluids. Judge Vanek concluded that the baby was murdered by an overdose of digoxin - though he found there to be no evidence that Susan Nelles was the perpetrator; indeed, in his view the evidence pointed against that conclusion.

Two things seem to have brought about a change of mind in the experts who appeared before me. It is now thought, at least by Dr. Hastreiter, that the possible presence of fecal matter from the bowel, which was cut at autopsy, rather than the above-mentioned edema and ascitic fluids, may have increased the digoxin level in the pelvic cavity. Secondly, Mr. Cimbura did some experiments in co-operation with Dr. Phillips on post-mortem blood from fourteen patients. He took digoxin levels on blood from the heart and the sagittal sinus and compared them to the levels found in the pelvic cavities both at autopsy and three hours later. Of the fourteen patients tested, the readings for gutter-blood (blood from the pelvic cavity) for all were reasonably similar to the heart and sagittal sinus - ranging from slightly smaller to double the amount - except in one sample where the readings at autopsy and three hours after were 169.6 ng/ml and 17.7 ng/ml respectively, compared to 9.9 ng/ml (heart) and 4.3 ng/ml (sagittal sinus); the figure of 169.6 ng/ml which looks like a mathematical error was rechecked several times and was found to be accurate. Drs. Hastreiter and Kauffman, because of this example, lost much faith in the 72 ng/ml reading. Dr. Hastreiter, however, felt there was a fair probability of digoxin overdose based on the child's clinical course alone. Dr. Mirkin, on the other hand, argued that an aberrant reading in only one example out of twenty-six (second pelvic cavity samples were omitted on two patients) bolsters the validity of the 72 ng/ml reading. Dr. Nadas, in his report to Atlanta, rated the baby's death unexpected, consistent with her clinical condition, and consistent with special concern regarding digoxin toxicity.

(24) FRANK FAZIO

Diagnosis

This thirty-two day old boy suffered from coarctation of the aorta opposite the ductus arteriosus which was patent. There was also an atrial septal defect and the mitral valve was somewhat obstructed. He also had a congenital disease (ichthyosis) resulting in hardening of the skin. His circulatory system was relatively normal.

The problem was that blood from the lungs had difficulty getting to the left ventricle through the mitral valve and would tend to go through the septal defect to the right atrium. Also, the blood going through the aorta would be obstructed by the coarctation.

Prognosis

The views of the cardiologists were as follows: Dr. Rowe - high risk death, Dr. Nadas - poor, Dr. Hastreiter - 7 (out of 10).

Course of Treatment

On admission to the Hospital at two days of age, the baby was cyanotic and had an enlarged liver indicative of congestive heart failure. Prostaglandin therapy was prescribed to keep the ductus open. On January 7, 1981, at age four days, he underwent surgery to repair the coarctation and for ligation of the ductus. His post-operative course was complicated by continued congestive heart failure, problems in feeding and breathing, and enterocolitis (inflammation of the intestines). He was treated with digoxin and diuretics. Although he returned to the ward on January 12, five days after surgery, he was back in the I.C.U. on January 30 because of a deteriorating respiratory condition. He returned to the ward again on February 2. While the transfer note indicated he was stable, his vital signs were irregular; he was described as generally in poor condition, and there were signs of sepsis. He also experienced bouts of apnea, rigidity in the limbs (seizures), and tachycardia. On February 3 he seemed somewhat better with a regular heart rate, improved respirations, and other stable vital signs.

(FRANK FAZIO)

Terminal Events

In the early morning of February 4, at approximately 3:45 a.m., the child became suddenly bradycardic, went into ventricular fibrillation alternating with bradycardia and what were described in the chart as "vast" heart rate irregularities, and suffered a cardiac arrest from which he could not be resuscitated. He was pronounced dead at 4:45 a.m.

Autopsy

There was no autopsy; permission was denied.

Toxicology

Several digoxin levels were taken in early January and produced readings all within the therapeutic range. The last reading taken yielded a level of 1.5 ng/ml on January 26, the week before death. No post-mortem tests were conducted.

The Experts

The terminal events, according to all the experts, were consistent with digoxin toxicity. On the other hand, they were not only consistent with his clinical condition, but the death was expected according to Dr. Nadas, who also rated it in his report to Atlanta, consistent with both his clinical condition and digoxin toxicity. Dr. Rowe indicated that the staff cardiologists at the Hospital attributed death to sepsis and congestive heart failure. He shared this view. Dr. Hastreiter thought digoxin toxicity unlikely. Dr. Fay said that, in his opinion, it would be irrational to ascribe his death to anything but natural causes. Dr. Mirkin, considering the suddenness of the terminal events, the bradycardia, and the ventricular fibrillation, thought digoxin toxicity possible.

(25) BRUCE FLORYN

Diagnosis

This nineteen year old boy was admitted to the Hospital for the last time on January 27, 1981. His problem was not an anatomic deformity but an inadequacy of the conduction system. As referred to earlier, the conduction system works by sending signals from the sinus node, the natural "pacemaker" of the heart, across the atria and via the atrioventricular node through the septum to the ventricles. This boy suffered from an interruption of that passage which is known as "congenital heart block", with the result that the ventricles were beating at only about half the rate of the atria. The gravity of this condition depends on how well the lower chambers of the heart can compensate for the higher rate of beating in the atria - the greater the discrepancy in rates between the two areas, the greater the danger to the patient.

In 1974, he had had a pacemaker installed to correct this problem but nevertheless suffered from advanced heart muscle disease (cardiomyopathy) which resulted in congestive heart failure and chronic liver disease. As his muscle disease worsened, the mitral valve in the heart failed to shut properly, and the left side of his heart became enlarged.

Prognosis

The views of the cardiologists were as follows: Dr. Rowe did not give a written prognosis for the boy but indicated in evidence that his condition was terminal; Dr. Nadas - poor, Dr. Hastreiter - 9 (out of 10).

Course of Treatment

There really was very little that could be done for the boy except to make him comfortable. He was considered for a heart transplant but he was ultimately rejected for the procedure. A "Do Not Resuscitate" order was in place. He was receiving digoxin and furosemide, but the medications were not controlling his heart failure. He became increasingly disoriented and constant nursing care at night, followed by shared nursing care by day, was provided in the two days prior to his death.

(BRUCE FLORYN)

Terminal Events

His last days in the Hospital were marked by gradual deterioration. On February 6, he had a fall and became drowsy and unresponsive. On February 7 at 6:15 a.m., a doctor was called because of weak respirations, increased cyanosis, and fixed and dilated pupils. He exhibited none of the symptoms usually manifested in digoxin toxicity cases. He was pronounced dead at 6:20 a.m.

Autopsy

At autopsy there were no unexpected findings. Death was attributed to cardiac failure secondary to cardiomyopathy.

Toxicology

The boy's last ante-mortem digoxin level, taken approximately one week before death, was 2.1 ng/ml, within the therapeutic range. Fixed heart tissue and frozen heart muscle were tested post-mortem. The heart tissue, tested only by RIA, produced results within the therapeutic range as did the heart muscle specimen after RIA plus HPLC and RIA.

The Experts

There was general agreement that the terminal events were inconsistent with digoxin toxicity. All the experts who expressed an opinion attributed death to his cardiac condition. Dr. Nadas, in his report to Atlanta, rated the boy's death expected, consistent with his clinical condition, and inconsistent with digoxin toxicity.

(26) JENNIFER THOMAS

Diagnosis

This nine day old baby girl suffered from a hypoplastic left heart and a very narrow aorta with an atretic (closed or almost closed) aortic valve. There was also an atrial septal defect and a small mitral valve. In the result, blood from the right ventricle was pumped to the lungs in the ordinary way, but on returning to the left side could not exit to the aorta, and instead most of it went through the atrial septal defect to mix with the blue blood returning from the body. The only way any oxygenated blood could circulate through the body was through the ductus arteriosus which was still patent on her admission to hospital. In the usual case, such a condition leads only to death.

This infant was noted as having mild dysmorphic features.

Prognosis

The views of the cardiologists were as follows: Dr. Rowe - inevitable death, Dr. Nadas - poor, Dr. Hastreiter - 10 (out of 10).

Course of Treatment

This baby was admitted to the Hospital on February 11, 1981, the day before she died, with a heart murmur which had intensified since the day of her birth, a rapid respiratory rate, and an enlarged liver. As it was vital to keep the ductus open, the child was prescribed prostaglandin therapy. Unfortunately, she developed some side effects and the dosage had to be reduced. The intention was to operate but it was recognized that in light of the baby's condition and her age, the surgery could only be described as "heroic". Shortly after her admission she developed an irregular heart rate and digitalization was commenced and diuretics prescribed. She was placed on both apnea and cardiac monitors for close observation. In the last nursing note, the baby was described as stable and feeding eagerly.

(JENNIFER THOMAS)

Terminal Events

The operation was scheduled for February 12, the day after admission. At 3:00 a.m. of that day her apex suddenly became irregular and she quickly experienced arrhythmias, bradycardia, shallow respirations, and ventricular fibrillation, and cardiac arrest followed. Attempts to resuscitate failed and she was pronounced dead at 3:38 a.m.

Autopsy

The autopsy confirmed the diagnosis and in the Final Autopsy Report death was attributed to "congenital heart disease - hypoplastic left heart - recent subendocardial infarct" in the heading. It was noted, however, that the ductus was still patent. No specific cause of death was given.

Toxicology

There were no blood levels taken, ante-mortem or post-mortem. Post-mortem levels were taken on fixed heart and lung tissues. The results from the heart tissue, in part tested by RIA only, and in part tested by RIA plus HPLC and RIA, were in the therapeutic range; those from the lung, tested by RIA plus HPLC and RIA, were in the toxic range. Mr. Cimbura estimated that levels in both would have been higher at death than when recorded.

The Experts

Dr. Nadas, in his report to Atlanta, rated the baby's death as unexpected and consistent with both her clinical condition and digoxin intoxication. Dr. Rowe did not regard the baby as at imminent risk of death. Notwithstanding the autopsy finding that the ductus was still patent, he attributed death to the closing of the ductus. He said that experiments with animals have shown that the ductus may remain moderately wide open and still permit no blood to get through. Drs. Fay and Hastreiter entertained only a "low" suspicion of digoxin toxicity. Dr. Hastreiter went so far as to say it was unlikely.

(27) DAVID LEITH

Diagnosis

This forty-two day old boy suffered from a complete atrioventricular defect, that is, there was essentially no wall or septum between the two sides of the heart. There was a common atrioventricular valve in place of the normal tricuspid and mitral valves. In addition, there was a hypoplastic left ventricle, obstruction of the aorta, and a patent ductus arteriosus. He, as a result, suffered from many complicated circulatory problems.

Prognosis

The views of the cardiologists were as follows: Dr. Rowe - inevitable death, Dr. Nadas - poor, Dr. Hastreiter - 6 (out of 10).

Course of Treatment

There was no real hope of corrective surgery for his major defect. Palliative surgery was considered, but was not thought feasible until the baby was older. The only things that could be done were a repair of the coarctation of the aorta and a ligation of the ductus performed on February 2, 1981. After the operation, he continued in heart failure, did not feed well, and experienced seizures. At the referring hospital, he had received digoxin and furosemide, and the drugs were continued at the Hospital. His course, despite treatment for his congestive heart failure, was progressively downhill. A "Do Not Resuscitate" order was in place.

Terminal Events

The infant began gravely deteriorating at 4:20 a.m. on March 6. He became cyanosed, distressed, and experienced fast breathing. Various palliative measures were taken to no avail. At 10:25 a.m. on March 6, he died in his mother's arms.

Autopsy

There was no autopsy.

(DAVID LEITH)

Toxicology

The baby had a slightly elevated digoxin level (2.8 ng/ml) four days before death. The last level taken on the day before death was 2.1 ng/ml. No post-mortem levels were taken.

The Experts

Dr. Rowe described his final course as one of steady decline. Most experts agreed that his terminal events were inconsistent with digoxin toxicity and entirely consistent with his clinical condition. Dr. Nadas, in his report to Atlanta, rated this baby's death expected and consistent with both his clinical condition and digoxin toxicity. The death was expected and all experts agreed that it was natural.

(28) COLLEEN WARNER

Diagnosis

This twenty week old girl was admitted to the Hospital at 7:36 p.m. on March 6, 1981, and died very early the next morning.

She had an unusual combination of malformations including endocardial fibroelastosis (thickening of the ventricular wall), cardiomyopathy (disease of the heart muscle), a concomitant rapid heart rate, a large ventricular septal defect, an undersized right ventricle, and a grossly oversized left ventricle. She had failed to thrive, was in respiratory difficulty, and also in congestive heart failure. Because of her defects, the blood entering both the pulmonary artery and the aorta was mixed and the pumping activity of the left ventricular cavity was inhibited.

Prognosis

The views of the cardiologists were as follows: Dr. Rowe - high risk death, Dr. Nadas - poor, Dr. Hastreiter - 6 (out of 10) for the cardiac ailment, but he noted that the infant also suffered from lung disease.

Course of Treatment

On admission, she was diagnosed as having sinus tachycardia with ventricular hypertrophy (overdevelopment); the ventricular septal defect was not initially suspected. She was treated with furosemide and a digitalizing dose of digoxin in the Emergency Room and given further intravenous digoxin after transfer to the ward. At this time, she still suffered from tachycardia and respiratory distress. The baby was constantly crying and very irritable, her colour dusky, but her heart rate remained steady, and she was thought to be improving.

Terminal Events

At 3:00 a.m. on March 7 she suffered a sudden attack of bradycardia and her blood pressure fell. Within five minutes her heart rate was hardly audible and a Code 25 was called. At 3:45 a.m., she was pronounced dead. Her terminal events were marked by ventricular flutter (a form of rapid ventricular tachycardia), bradycardia, and junctional rhythm.

Autopsy

The autopsy revealed the large ventricular septal defect and the small right, and large left, ventricles. She also suffered from extreme cardiomegaly. The tricuspid valve was slightly underdeveloped. There was evidence of inflammation of the lungs and viral pneumonitis. The heading of the Final Autopsy Report attributed death to "ventricular septal defect: cytomegalovirus inclusion disease" (a serious viral disorder).

Toxicology

There were no ante-mortem levels taken. Digoxin levels were taken on fixed heart tissue, and the results obtained using RIA plus HPLC and RIA were in the therapeutic range, although those of the left ventricle were within the overlap range (which includes both toxic and therapeutic levels). Mr. Cimbura tested the preservative solution by both RIA and GC/MS and estimated that the digoxin level in her fresh heart tissue would have been in the overlap range.

The Experts

Dr. Nadas, in his report to Atlanta, found the baby's death unexpected but consistent with both her clinical condition and digoxin toxicity. Dr. Rowe did not consider the possibility of digoxin toxicity at the time of death. He thought the cause of death was likely related to her endocardial fibroelastosis. Dr. Rose, who was ward chief at the time, was worried about digoxin sensitivity contributing to the terminal events because the child's particular condition of fibroelastosis predisposed her to digoxin toxicity from even a normally therapeutic dosage. Dr. Rose, however, was satisfied as to a natural cause of death after viewing the autopsy report. Dr. Fay thought there was only a low chance of digoxin toxicity; Dr. Hastreiter thought a massive overdose was a fair probability, largely because of the suddenness of the terminal episode.

(29) JORDAN HINES

Diagnosis

This twenty day old baby boy was found by his mother coughing and choking in his crib some four days before his death. She picked him up and he quickly settled down. His colour was normal and his breathing seemed to her to be regular as well. She arranged for the baby to be examined by a physician who reported that the baby had gained weight and was developing well. Later that afternoon, she noticed that his breathing was shallow and he appeared dusky in colour. She picked him up, shook him, and he responded immediately, but he again turned dusky and his breathing became shallow. She and the infant's father then took him to the North York General Hospital. On the way, there was another episode of shallow breathing and a change in colour, averted when the mother stimulated the baby.

At the North York General Hospital, he suffered similar episodes including several periods of apnea. At about midnight on March 5, 1981, he was transferred to the Hospital for Sick Children. At the referring hospital, the final diagnosis was: "sick sinus syndrome with bradycardia and tachycardia probably due to some form of congenital heart disease". "Sick sinus syndrome" was never clearly defined for us and it does not appear in any of the common medical dictionaries. It does appear to be a disturbance of the sinus node (the natural pacemaker of the heart) which results in an electrical instability in the conduction system. Some of the symptoms of this disorder are arrhythmia, bradycardia, and tachycardia.

Prognosis

The views of the cardiologists were as follows: Dr. Rowe - high risk death, Dr. Nadas - guarded, Dr. Hastreiter - 3 (out of 10).

Course of Treatment

At the Hospital the initial diagnosis was infection or sepsis and possibly pneumonia. He was experiencing a fluctuating heart rate but there was no cardiac murmur; on admission he was described as lethargic and his colour was good. Antibiotics were prescribed. An echocardiogram was conducted and revealed a structurally normal heart. He was placed on both an apnea and a cardiac monitor for observation. Cultures taken to test for sepsis were negative.

Obviously no cardiac surgery was planned. On March 6, one or two mild apneic episodes were noted and he also had bouts of bradycardia and tachycardia. At times he was slightly cyanotic but did not appear to be otherwise in distress. Dr. Kobayashi, a resident who was in attendance on March 7 and 8, found nothing untoward and heard no report from the nurses concerning any problem in the child's condition. The infant's vital signs were normal; there was no fever, indeed, no cause for concern. He was extremely stable when Dr. Kobayashi retired for the night between 1:00 and 2:00 a.m. on March 8. The baby was still on the two monitors.

Terminal Events

At 3:00 a.m. on March 8 the baby vomited, but fed a little, and went back to sleep. He was in no distress with a regular apex. At 4:00 a.m. he experienced tachycardia, but the apex was still regular, and he was not in distress. At 4:10 a.m. his cardiac monitor suddenly went off, followed immediately by his apnea monitor, and he could not be aroused when shaken. He then went into cardiac arrest and cardiopulmonary resuscitation was started. A long resuscitation effort followed, in which more than once there was a return to normal sinus rhythm only to be followed repeatedly by ventricular fibrillation, and ultimately bradycardia and occasional junctional rhythm. The baby was pronounced dead at 6:43 a.m.

Autopsy

The autopsy was conducted by Dr. Becker who confirmed the normal structure of the heart and the absence of any extracardiac malformation. He found no evidence of infection. He did, however, find evidence of chronic hypoxia, and also the following specific findings which he said (and he is recognized as a world-renowned authority on the subject) were indicative of "missed-SIDS" - sudden infant death syndrome, (a) gliosis (scars in the brain stem), (b) extra-medullary hematopoiesis, that is, the forming of red blood cells in parts of the body other than the bone marrow, (c) persistence of brown fat, i.e., the fetal fat that is usually lost after birth, and (d) thickening of the pulmonary arterioles, that is, thickening of small arteries in the lung. These findings according to Dr. Becker are findings associated with missed-SIDS which, as I understand it, is a term used by pathologists to describe a death resulting from SIDS in which there was a previous episode of a similar nature which did not result in death. Dr. Becker put it in the autopsy report thus:

> This pathologic evidence, in conjunction with the clinical history, makes the diagnosis of a missed-SIDS a possibility. However, this does not explain the arrhythmias and further conclusions will have to await examination of the conducting system.

Dr. Becker listed as the principal finding in the heading of the Final Autopsy Report: "query sudden infant death syndrome." In combination, these comments appear to indicate some hesitation or qualification of the finding, but Dr. Becker in evidence said he was certain of the diagnosis, and was not very concerned about the arrhythmias. He did concede, however, that there was a possibility that something in the conduction system caused or contributed to his death. I might just add here that no examination of the conduction system - a very long, complicated procedure - was ever carried out.

Toxicology

The child was not prescribed digoxin. There was heart and lung tissue available, fixed in preservative solution following autopsy. The body was exhumed on December 8, 1981 (shortly before the beginning of the Preliminary Inquiry), at the instigation of the baby's father who became very suspicious about the cause of his son's death. At that time, tissue specimens from the thigh and liver became available. All tissues were tested by RIA plus HPLC and RIA (save for part of the fixed heart tissue and the exhumed liver tissue) and all of them contained digoxin. The fixed heart tissue levels were in the therapeutic range. Mr. Cimbura estimated that the probable level in fresh heart tissue would have been within both the therapeutic and toxic ranges. The fixed lung reading was also within the overlap range. The exhumed liver tissue was well above the therapeutic range but, as noted, was tested by RIA only.

The presence of digoxin whether in therapeutic or toxic levels is of course very significant as no digoxin was prescribed.

Medication Error

As in the cases of Stephanie Lombardo and Jesse Belanger, Dr. MacLeod suggested that a dose of digoxin administered accidentally to the baby at any time following his admission to the Hospital could account for his post-mortem levels. Dr. Spielberg agreed. However, the only drugs prescribed for this child were the antibiotics ampicillin and gentamycin. On the evidence, the possibility of confusing these drugs with digoxin is most unlikely. Particularly in the case of ampicillin, which comes in a powdered form and has to be diluted in solution, it would be almost impossible to make that mistake. It is of course, always possible that digoxin was given to this baby which should have been given to some other patient but there is no evidence to support that theory. Dr. Kauffman thought a medication error unlikely, but in any event was of the view that a single dose (in a maintenance or loading amount) would not account for the levels. Dr. Hastreiter agreed that one accidental maintenance dose would be insufficient but suggested that one digitalizing or loading dose might produce the recorded levels. However, he thought such an error was unlikely in view of the medications the baby was receiving. Dr. Mirkin also thought a medication error unlikely.

The Experts

There are essentially three possible causes of death: (1) SIDS, (2) sick sinus syndrome, and (3) digoxin toxicity. The experts were most decidedly not in agreement; while nearly all found the death consistent with digoxin toxicity, that was strangely not the conclusion of Dr. Nadas who, in his report to Atlanta, rated this infant's death expected, consistent with his condition, and inconsistent with digoxin toxicity. The infant possessed two criteria either of which placed him in the Atlanta Report Category A: Dr. Kauffman's digoxin score of 3 and Dr. deSa's conclusions discussed below. Dr. Becker, as we have seen, reached a decision at the time of the autopsy in favour of missed-SIDS and he was supported by Dr. Bain and almost all the other Hospital doctors after they had read his report. They all, however, were concerned about the toxicologic data and conceded that if the readings were valid, digoxin toxicity may have played a part in the death.

Most of the experts, including Dr. Rose who was the staff cardiologist on call at the Hospital during the baby's resuscitation, confirmed that deaths attributable to SIDS do not usually occur in hospitals where sophisticated monitoring techniques are available. The reports published in the medical and scientific literature indicate that SIDS is not commonly a cause of death in children under thirty days of age, although of course there are exceptions.

It should perhaps be noted as well that the death of Jordan Hines caused considerable concern amongst members of the nursing staff who had been involved in his care. At the time of his death, they regarded it as very unexpected and inconsistent with what they had thought to be a stable and relatively well child. Certainly no one felt him to be at imminent risk at the time that he died.

All the outside doctors rejected SIDS outright as the cause of death. Dr. deSa defined SIDS as a diagnosis by exclusion (a diagnosis made when no other possible cause of death exists), and doubted that there were specific pathologic findings related to it. This was one of the three cases in which Dr. deSa felt the autopsy findings did not completely explain the death nor the acute mode of death. He considered the four "specific" findings of Dr. Becker on autopsy consistent with chronic hypoxia. Dr. Hastreiter also agreed that hypoxia could have caused the autopsy findings. He also thought the arrhythmias were inconsistent with the diagnosis of SIDS. He thought the child may indeed have suffered from sick sinus syndrome but noted that death from that syndrome was unusual. Drs.

(JORDAN HINES)

Hastreiter, Fay, and Kauffman all thought death from digoxin toxicity much the most likely cause. Dr. Kauffman found the child's course in hospital completely inconsistent with SIDS. Dr. Mirkin also rejected SIDS in favour of digoxin toxicity as the probable cause of this baby's death.

(30) BARBARA GIONAS

Diagnosis

This forty-six day old baby girl suffered from multiple ventricular septal defects, a coarctation of the aorta, a patent ductus arteriosus, and an atrial septal defect. As a result, her circulatory system was abnormal, characterized by the shunting of large amounts of blood from the left to the right side of the heart. There were additional problems not immediately diagnosed including a thickness of the upper septum indicating hypertrophic (enlarged) muscle disease. There were also some mild dysmorphic features although the first chromosomal study showed no abnormality.

Prognosis

The views of the cardiologists were as follows: Dr. Rowe - high risk death, Dr. Nadas - guarded, Dr. Hastreiter - 8 (out of 10).

Course of Treatment

At birth, a heart murmur was detected and transient tachypnea was observed as was an enlarged liver. She was assessed as having congenital heart disease and was transferred to the Hospital the next day where a cardiac catheterization was performed. A ligation of the ductus and repair of the coarctation were carried out at four days of age. There was still, however, persistent heart failure believed to be caused by massive amounts of blood passing through the septal defects producing an overflow in the pulmonary artery. The latter was banded in a second operation at four weeks of age. There was some improvement thereafter, but she remained in congestive heart failure, accompanied by tachypnea and continued to have an enlarged liver. She had to be mainly fed by naso-gastric tube as of March 2 and generally failed to thrive. She was treated with digoxin and diuretics.

On March 7, at approximately 1:00 p.m., her heart rate went up and she vomited twice. The attending resident listed as his impression at the time: digoxin toxicity, congestive heart failure, and hyponatremia (deficiency of sodium in the blood). He ordered that the next dose of digoxin be withheld, that she be placed on a fluid restriction, and that a chest X-ray and electrocardiogram be performed. The electrocardiogram revealed atrial flutter and heart block, whereupon the resident's primary impression was revised to digoxin toxicity. A digoxin level was ordered and the drug was to be withheld for forty-eight hours. The level when measured was 1.2 ng/ml, well within the therapeutic range. On March 8, the day before her death, the nursing notes reveal an improvement in her condition. She was then described as stable, settled, and restful. That night, however, her heart rate was consistently irregular; she again was restless and very difficult to settle.

Terminal Events

On March 9, at approximately forty-five minutes past midnight, the child became increasingly restless and started to perspire; within minutes the baby's heart rate started to fall and she became bradycardic. A doctor was called by a Code 23 and she gave furosemide and atropine, but cardiac arrest followed and resuscitation efforts failed. Death was pronounced at 1:45 a.m.

Autopsy

Although consent was given for an autopsy, and autopsies are routine upon consent, no autopsy report is in the baby's chart and no record of an autopsy could be found.

Toxicology

The child was on digoxin and there was some concern about suspected toxicity in spite of digoxin levels in the therapeutic range. The prescribed dose was reduced on March 3 and, as noted above, held on March 7 and never restarted. The highest ante-mortem levels were 2.3 ng/ml on February 3 and February 5; there were no post-mortem blood level readings.

The only post-mortem tests were carried out on exhumed tissue specimens: namely, on heart, liver, lung, and muscle tissue, and on stomach and bowel contents. All tests were conducted using RIA plus HPLC and RIA. While the heart tissue concentrations were in the overlap area (within both therapeutic and toxic ranges), and the liver and lung concentrations were in the toxic range, for the reasons I have given the results must be considered inconclusive.

The Experts

Dr. Nadas, in his report to Atlanta, rated the death unexpected but consistent with her condition. He also stated special concern regarding digoxin toxicity. Dr. Kauffman did not believe the Hospital course of the baby was consistent with digoxin toxicity but believed that her death was. Dr. Fay and the Hospital doctors did not think that digoxin was involved in her death. Dr. Hastreiter, however, thought it was quite possible. Dr. Rose, the cardiologist responsible for the baby, ascribed her death to her many problems: respiratory illness, unstable temperature, electrolyte imbalance, anemia, and heart disease. Dr. Kobayashi, the pediatric resident who cared for her until 11 p.m. March 7, considered her condition stable and her death a surprise.

(31) MICHELLE MANOJLOVICH

Diagnosis

This nine month old baby girl suffered from pulmonary atresia, a hypoplastic (underdeveloped) right and an enlarged left ventricle, and an atrial septal defect. In addition, her tricuspid valve was smaller than normal. As a result, her blue blood would go through the septal defect to the left chamber and out to the aorta. The only way for blood to reach the lungs and be oxygenated was by the ductus arteriosus, and then only so long as it remained patent. As the ductus closes the patient becomes progressively more cyanosed.

Prognosis

The views of the cardiologists were as follows: Dr. Rowe - high risk death, Dr. Nadas - poor, Dr. Hastreiter - 8 (out of 10).

Course of Treatment

The baby was originally treated with the drug prostaglandin to keep the ductus open. On June 24, 1980, a valvotomy was performed to allow some blood to enter the pulmonary artery and a shunt was constructed between that artery and the aorta. At the conclusion of the infant's first stay in the Hospital, she was sent home with instructions to her parents to continue the administration of the necessary drugs including digoxin.

On January 6, 1981, she was reassessed and a number of symptoms indicating congestive heart failure were observed. She was readmitted and underwent a second cardiac catheterization in the Hospital on January 19, returning home once again on January 20. The catheterization revealed that her shunt was functioning although there was considerable shunting of blood between her two atria, and the pressure in her right ventricle was abnormal. The baby returned to the Hospital on February 2. In order to try to relieve the situation, a valve and patch were inserted to enlarge the pulmonary artery and the right ventricle and the original shunt was also enlarged. She had a stormy post-operative course, showing signs of hepatitis and acute liver failure, with continuing congestive heart failure, but she made gradual improvement and was transferred back to the cardiac ward from the I.C.U. on March 2. She suffered a respiratory set-back on March 4 and was returned to the I.C.U. and put on a ventilator. On March 7 she was returned to the ward and over the next few days was again showing gradual progress towards recovery. She was treated with digoxin and diuretics.

Terminal Events

During the long night shift on March 11 the baby was irritable and had difficulty feeding although her vital signs were stable. That day she had been stable and comfortable and, in fact, had been thought to be improving. In the early morning of March 12, at approximately 2:30 a.m., she suddenly became bradycardic and went into cardiac arrest. Resuscitation efforts failed. She was pronounced dead at 3:35 a.m.

Autopsy

There was no autopsy; parental consent was denied.

Toxicology

There had been some difficulty in maintaining proper therapeutic levels of digoxin earlier in February. Two "hold" orders were issued in the last two weeks of the baby's life. Her digoxin level on March 11 (the day before her death) was 2.2 ng/ml. No concern was expressed by Dr. Rowe regarding any of the baby's ante-mortem digoxin levels which at no time exceeded 3.3 ng/ml. A sample described as "amber coloured fluid" was tested at the Centre of Forensic Sciences by RIA plus HPLC and RIA and did not reveal any digoxin. There is no evidence to suggest that this was a post-mortem sample of blood. The child was not exhumed and no tissue specimens were tested.

The Experts

Dr. Nadas, in his report to Atlanta, found the baby's death to be unexpected but consistent with the child's clinical condition. He also thought it consistent with special concern regarding digoxin toxicity. Drs. Rowe and Costigan thought the baby died from a respiratory problem, possibly an aspiration of vomit or food similar to the incident on March 4. Some support for this theory is found in the fact that the child's mouth was found to be full of food, with some on the pillow, at resuscitation. The other Hospital doctors were inclined to agree, although nothing could clearly be established in the absence of an autopsy. Dr. Rose considered the infant's heart problem to be lethal and her death not unexpected. Dr. Fowler described her prognosis as ominous. All the Hospital doctors agreed that the onset of her terminal events had been sudden. Dr. Hastreiter thought there was a fair probability of digoxin overdose.

(32) KEVIN PACSAI

Diagnosis

This twenty-five day old boy, like Jordan Hines, (and Lauretta Heyworth at the beginning of her life), had a structurally normal heart. He was admitted to a hospital in Hamilton originally, where he was found to be suffering from arrhythmias and heart failure. While there, he suffered an episode of serious shock and almost died, but his condition improved with treatment. The diagnosis by his doctor in Hamilton was of paroxysmal atrial tachycardia (periods of excessively rapid heart beat), a condition which usually responds favourably to appropriate treatment. The child had also been experiencing high potassium levels. He was transferred to the Hospital for Sick Children on March 11, 1981, for investigation because of suspicion of some congenital dysfunction of the conduction system.

Prognosis

The views of the cardiologists were as follows: Dr. Rowe - low risk death, Dr. Nadas - good, Dr. Hastreiter - 2 (out of 10).

Course of Treatment

On admission to the Hospital on March 11, the baby seemed normal and stable. His condition was then of no concern to Drs. Kantak and Kobayashi who examined the baby following his admission. His potassium level, measured that night, was 3.9 mEq (milliequivalents), within the normal range. That drug was not prescribed at the Hospital. Various examinations and tests were carried out but no cardiac catheterization was planned. Digoxin had been administered in Hamilton and one dose was given at the Hospital, as were diuretics.

Terminal Events

A few hours after admission, the baby suddenly developed alternating tachycardia and bradycardia. The nurse in attendance (Susan Nelles) discovered his deterioration on returning from the resuscitation effort concerning Michelle Manojlovich. The nurse noticed in particular that at 4:00 a.m. March 12 his feeding habits had entirely changed; from an active baby, he had become lethargic and uninterested in feeding. His cardiac monitor showed alternating bradycardia and tachycardia and occasional heart block. His respirations were shallow, his blood pressure plummeted, and his extremities were blue. She had some difficulty persuading the cardiac fellow and the pediatric resident on duty of the urgency of this child's problem. Dr. Costigan, the senior resident, who had seen the baby earlier when his condition did not seem serious, was notified. He attended on the ward, verified the existence of serious arrhythmia and heart block, and made arrangements to transfer the child to the I.C.U. On leaving the ward to go to the I.C.U., the baby again experienced bradycardia and a brief period of apnea but responded to stimulation. In the I.C.U. the baby suffered further episodes of bradycardia and heart block. A potassium level was taken and resulted in an elevated reading of 9 mEq. This sample however, was slightly "hemolyzed" (indicating a breakdown of the red blood cells likely to lead to an artificially high reading) and a second sample was tested, also resulting in an elevated level (7.7 mEq). Dr. Costigan took steps to lower the level and, suspecting digoxin toxicity, issued a "hold" order on that drug. The child maintained sinus (normal heart) rhythm for at least an hour, but at 8:45 a.m., with Dr. Costigan still in attendance, the baby became apneic with severe bradycardia and ventricular fibrillation. Cardiopulmonary resuscitation was undertaken and was successful for a while, but the child reverted to ventricular tachycardia and his heart rhythm continued to be irregular. Eventually the child was pronounced dead at 10:10 a.m.

Autopsy

This was no routine autopsy. First of all, Dr. Fowler caused the death to be reported to the Coroner, not so much because of a concern regarding the cause of death, but more because of the serious reaction of the baby's father to his death. The reporting of deaths to the Coroner was not in itself unusual. The deaths of babies

Woodcock, Velasquez, and Dawson had all been so reported. But Dr. Costigan continued to be worried about the high potassium level. He wondered if his efforts to reduce it had caused the digoxin to have an enhanced effect. He was puzzled as to how the potassium level had risen from 3.9 mEq (on the night of the baby's admission) to 7.7 mEq in less than twelve hours when the drug had not been administered. Digoxin is known to have a greater pharmacologic effect on the heart, that is, to be more toxic, in cases where a patient's potassium levels are abnormally low. A blood sample had been taken ante-mortem, at about 6:30 a.m. on March 12, for purposes of a complete blood count. Dr. Costigan obtained this sample from the Haematology Department and gave it to Dr. Ellis, the biochemist, for digoxin testing. Dr. Costigan had confirmed with Dr. Ellis beforehand that this sample taken for another purpose would be suitable for a digoxin test.

Dr. Cutz was the supervising pathologist at the autopsy performed under a Coroner's Warrant on March 13. There were no abnormal heart findings or significant pathologic findings of any sort. He himself, for the first time in his career, ordered a post-mortem blood test for digoxin after reading the child's medical chart. He wrote the Final Autopsy Report after receiving the results of the digoxin tests and ascribed death to digoxin toxicity because of the high ante-mortem and post-mortem digoxin levels.

Toxicology

At the referring hospital in Hamilton, a digoxin level taken on March 9 had been 1.8 ng/ml, well within therapeutic limits. The test ordered by Dr. Costigan (on the ante-mortem sample taken at about 6:30 a.m. on March 12) resulted in a level of greater than 10 ng/ml. The sample had been preserved with the addition of ethylene diamine tetra-acetic acid (a preservative of blood used in all such haematology samples). Dr. Ellis was, nevertheless, satisfied by tests undertaken by him that this substance would not affect the validity of a digoxin reading. It was only possible to dilute the sample once; the computer projection on the undiluted sample was 16 ng/ml; the computer projection on the first dilution was either 10.6 ng/ml or 21.2 ng/ml. Dr. Ellis was not sure from the digoxin test documentation which projection had in fact occurred, but in any event, he did not put much faith in computer projections. The actual level could not be ascertained on further dilutions because of an insufficient quantity of blood.

The post-mortem sample taken by Dr. Cutz was subjected to many tests at three different laboratories; first at the Hospital for Sick Children by RIA only in several dilutions conducted on two separate days. These resulted in a level of 26 ng/ml. Part of the unused portion of the sample was sent to Mount Sinai Hospital to be tested there and the answer (reached after considerable checking and reported to the Hospital within two weeks but not available to the Police for many months) was 112 ng/ml - also tested by RIA but with a different digoxin test kit which used different antibodies. Finally, a test at the Centre of Forensic Sciences, using RIA plus HPLC and RIA analysis, arrived at a level of 26 ng/ml. As we have seen, the post-mortem multiplier can be anything from one to five. We also know that the ante-mortem reading was greater than 10 ng/ml in this case. So, in this instance (if we ignore the anomalous Mount Sinai reading), it was not possible for the multiplier to be greater than 2.5. With the child's last scheduled dose being seven hours before onset, he would almost certainly have been at steady state with respect to that dose at death if only prescribed digoxin had been administered. A figure of 10 ng/ml is three to four times the normal therapeutic maximum level.

When Dr. Costigan received the digoxin level out test results (he believed it was Tuesday, March 17) he reported the matter to Dr. Fowler. The Chief Resident has a special relationship with the Chief of Pediatrics, and Dr. Costigan reported the matter to Dr. Carver as well. The latter, besides instructing Dr. Fowler to report the level to the Coroner, also instructed him to investigate the matter further, particularly to determine if there had been a prescription, transcription, or dosage error. None was found. Dr. Carver also caused the biochemistry department to check the concentration of digoxin in the oral preparation. It too was found to be correct.

Tissue specimens were tested also; concentrations measured in fixed heart tissue (part of which was tested using RIA plus HPLC and RIA) were found to be in the overlap area (within both therapeutic and toxic levels). Mr. Cimbura estimated that the tissue before fixing would have had a higher level of digoxin, but he could not say how much higher it would have been. Both fixed and frozen lung tissue were tested for digoxin, again using RIA plus HPLC and RIA. In both cases the levels were above those normally obtained in babies receiving therapeutic doses of digoxin.

It is clear that Kevin Pacsai's post-mortem digoxin levels in blood, although well above therapeutic levels, did not approach the heights found in the post-mortem readings of some of the other children, i.e., babies Estrella, Inwood, Miller, and Cook. They did, however, approach the levels of one Gary Murphy who died in the Hospital on April 23, 1983, and into whose death an inquest was conducted the following month.

Gary Murphy

> Gary Murphy was prescribed digoxin therapeutically. The last ante-mortem level was taken on April 3, 1983, and was well within the therapeutic range. The Hospital at that time was routinely checking digoxin levels at autopsy. The post-mortem readings were by this time calculated in nanomoles per millilitre (another form of digoxin level measurement), but when converted to nanograms, the reading on a sagittal sinus blood sample at the Hospital using RIA only was 18.7 ng/ml. Another post-mortem sagittal sinus blood sample was later measured at the Centre of Forensic Sciences where the reading was 18.9 ng/ml. In addition, a heart blood specimen tested at the Centre of Forensic Sciences yielded a level of 32.2 ng/ml. In spite of this, the report of the autopsy performed under a Coroner's Warrant attributed death to complex congenital heart disease. In contrast to the fresh autopsy tissue to be discussed later under Justin Cook, Gary Murphy's digoxin level in heart tissue was only 331.2-356.2 ng/g (the normal range in heart tissue of infants on digoxin is 49-975 ng/g).

(KEVIN PACSAI)

In considering the toxicologic data available on Kevin Pacsai, we must also consider whether the facts concerning Gary Murphy assist in determining the cause of Kevin Pacsai's death.

None of the experts attributed Gary Murphy's death to an overdose of digoxin. Dr. Spielberg and Dr. Kauffman suggested that his particular pathophysiologic condition, for reasons not fully understood in medicine, could account for the high levels. Certain factors existing in life could cause digoxin to unbind from tissue and be redistributed in the blood thus producing an artificially high level. Dr. Hastreiter disagreed; although he conceded there was no supportive evidence in the chart, he attributed the levels to pre-renal failure which would result in the inability to excrete digoxin in the normal way.

There is no question that there were marked differences between Gary Murphy's condition and that of Kevin Pacsai. Gary Murphy aged six months (as opposed to Kevin Pacsai aged twenty-five days) had a horrendous series of inoperable heart defects, suffered continually from hypoxia and cyanosis, and his condition was complicated by the absence of a spleen and by gastroenteritis. He was considered terminally ill (Dr. Hastreiter rated him 9-10 on his severity scale). None of this applied to Kevin Pacsai. Drs. Hastreiter, Kauffman, and MacLeod all considered the cases readily distinguishable. Only Dr. Spielberg saw a parallel. I cannot accept that latter view. The digoxin levels in Gary Murphy are very hard to explain but his condition was so different from that of Kevin Pacsai that the two cases are simply not comparable. The Coroner's jury found that Gary Murphy died of natural causes.

Potassium

I earlier noted that an abnormally low potassium level can predispose a patient to toxicity from a therapeutic dose of digoxin. In addition, however, there appears to be a consensus amongst the experts that elevated potassium levels are known to accompany elevated digoxin levels, although this does not always occur. It was also agreed that elevated digoxin levels can cause potassium levels to rise; what is unclear is whether the reverse is also true, that is, whether high potassium levels can cause digoxin levels to rise. Drs. Spielberg and MacLeod were of the view that such a phenomenon could occur. Dr. Spielberg, alone of all the experts, suggested that it might account for Kevin Pacsai's digoxin levels. However, neither Dr. Kauffman nor Dr. Hastreiter was aware of any empirical scientific data reported in the literature to support this theory. While there was therefore a difference of opinion as to whether the increase in the baby's potassium level could have caused the increase in his digoxin level all doctors agreed an increase in his digoxin level could have caused his potassium level to elevate.

The Experts

Almost all the doctors attributed the death of Kevin Pacsai, at least at the time, to digoxin toxicity. Dr. Cutz, as I have said, could find no other reason. He thought it unlikely that it was caused by a conduction system fault or that the high potassium level contributed. Dr. Bain thought the cause of death to be transient adrenal insufficiency resulting in a fatally high potassium level, which might also have been the cause of the original episode of severe shock which occurred in the referring hospital. Transient adrenal insufficiency is a very rare affliction and being transient may be undetectable, even at autopsy. Drs. Kauffman and Hastreiter saw no evidence to support the hypothesis of Dr. Bain that transient adrenal insufficiency had ever occurred. They felt that the baby's previous condition of shock would have accounted for his abnormal potassium readings seen in Hamilton. Drs. Mirkin, Hastreiter, and Fay, as well as Dr. Kauffman, had little doubt that digoxin toxicity was the cause of death. As noted, all the pharmacologists were agreed that his high potassium level on the day of death could have been caused directly by digoxin toxicity. As I have said, Dr. Spielberg thought the cause of death

(KEVIN PACSAI)

might be pathophysiologic in origin, as in the case of Gary Murphy. Dr. Kauffman disputed the pathophysiologic theory in the case of Kevin Pacsai whose condition was not like that of Gary Murphy in any way except in the post-mortem digoxin levels exhibited by each. Dr. Nadas, in his report to Atlanta, rated the infant's death unexpected, inconsistent with his clinical condition, and consistent with special concern regarding digoxin toxicity.

(33) KRISTIN INWOOD

Diagnosis

This eighteen day old baby girl suffered from a moderate coarctation of the aorta. There was a tubular hypoplasia (underdevelopment) of the arch of the aorta and a localized constriction opposite the ductus arteriosus which was patent. The result of this condition was an enlargement of the heart; the circulation, while normal, was slightly impaired by the coarctation. She also had slightly dysmorphic features. There was a concern that congenital rubella (German measles) syndrome might have caused the heart problems.

Prognosis

The views of the cardiologists were as follows: Dr. Rowe - low risk death, Dr. Nadas - poor, Dr. Hastreiter - 6 (out of 10).

Course of Treatment

The baby was to undergo cardiac catheterization on March 13, 1981 - two days after admission to the Hospital - to confirm the diagnosis as a prelude to surgery to repair the aorta. She died before either procedure could be carried out. On admission she was observed to be in heart failure; however, the nursing notes for March 11-12 indicate no apparent distress with vital signs normal, although the baby was feeding poorly on the evening of March 12. Furosemide was prescribed and given intravenously at 11:10 p.m. on March 12.

Terminal Events

At 2:00 a.m. on March 13, the nurse in attendance noted abnormalities of heart rate. A resident was called and furosemide was again given intravenously. Tachycardia developed and the baby became very irritable. At 2:30 a.m., the baby suffered cardiac arrest accompanied by bradycardia. She could not be resuscitated and was pronounced dead at 3:00 a.m.

(KRISTIN INWOOD)

Autopsy

At autopsy the responsible pathologist, Dr. Cutz, could find no single clear cause of death. He found, however, in addition to her cardiac problems, pulmonary problems which appeared to be resolving. In the Final Autopsy Report he also stated that electrical instability of the heart could have resulted from observed myocardial necrosis (death of heart tissue). This problem alone, in his opinion, could have caused the infant's death.

Toxicology

This was the child who received in error the dosage of digoxin intended for Kevin Pacsai (.02 mg (milligrams) instead of her own dose of .006 mg), early on March 12, the day before her death. A digoxin level taken at 9:00 a.m., three and one-half hours after the dose, resulted in a level of 2.6 ng/ml; this was the only known dose which she received at the Hospital. Digoxin was then withheld until her death. A further blood sample, thought to have been taken later on March 12, was tested at the Centre of Forensic Sciences. It resulted in a negative reading for digoxin after testing by RIA plus HPLC and RIA. A serum sample, taken at autopsy for another purpose, was tested for digoxin ten months later and showed a level of 491 ng/ml - much the highest level of any of the babies under review and perhaps the highest level ever recorded. It is, in fact, 2 1/2 times higher than the upper end of the toxic ranges quoted by Mr. Cimbura.

The purity of the sample and hence the validity of the level were attacked; indeed, the level was so high that Dr. Kauffman found it difficult to believe. There were suggestions that the sample had been frozen or refrigerated between autopsy and the time of testing, and also that at some time it had been heated. Dr. Mirkin said the fact of freezing and subsequent testing ten months later would not likely affect the validity of the result. Neither, in his view, would heating change the result, except perhaps to decrease the concentration of digoxin. Mr. Cimbura conducted experiments to discover the effect of heating on serum samples, later tested for digoxin, and found no noticeable effect. Dr. Ellis conducted a similar test on specimens taken at autopsy from Justin Cook and found no material difference in the concentrations measured after heating the samples. Drs. Mirkin and Kauffman noted that freezing was a common method of preserving serum samples and indicated that the likelihood of contamination, or other factors which would affect the concentration of digoxin, was remote. Dr. Kauffman recognized that the possibility of evaporation due to lengthy freezing or refrigeration of the sample could have the effect of artificially increasing the digoxin concentration present. He pointed out, however, that even if one assumed a tenfold increase in digoxin concentration caused by some storage problem such as evaporation, the actual concentration at death would still be at a toxic level.

Several fixed heart tissue specimens became available and were tested by the Centre of Forensic Sciences. From six such specimens, no conclusions could be drawn because, although the levels were the highest seen in the fixed tissue of any of the children under review, they were in both the therapeutic and toxic ranges for fresh autopsy tissue after testing by RIA plus HPLC and RIA. As mentioned above, the baby was already receiving digoxin prior to death which would naturally have resulted in positive tissue levels. Exhumed muscle tissue specimens were tested by RIA plus HPLC and RIA and resulted in levels exclusively in the toxic range. For the reasons given above regarding exhumed tissues, no definitive conclusions could be drawn from those levels either.

Medication Error

It is unlikely that the digoxin error at 5:30 a.m. on March 12 had anything to do with the death in light of the reading of 2.6 ng/ml less than four hours after the error. The suggestion was made that the furosemide administered between 2:00 and 2:30 a.m., prior to the cardiac arrest, might actually have been digoxin given inadvertently. Dr. Fay considered a digoxin-furosemide error unlikely since the colour of the two ampoules and the printing on them differ completely. He also stated that an error in which the equivalent volume of digoxin was given in place of furosemide would not, in itself, have caused death. Dr. Kauffman stated that an administration of digoxin, in the equivalent volume of furosemide reported to have been given, at the time it was given, would not have produced the post-mortem serum level found. The amount of furosemide reportedly administered was 3 mg which would be contained in .33 ml of the furosemide preparation. The digoxin contained in that volume of the adult digoxin preparation would be .08 mg. Dr. Hastreiter estimated that a minimum dosage of digoxin necessary to produce the post-mortem serum level would be 1.3 mg (if given about one hour prior to death) or .65 mg (if given immediately prior to death) which would have required the administration of three or two adult ampoules respectively of digoxin, an unlikely prospect as an accident.

The Experts

Dr. Nadas, in his report to Atlanta, found the death unexpected but consistent with both her clinical condition and digoxin toxicity. Dr. Rowe thought death was the result of the baby's clinical condition, although he conceded that if the toxicologic data were reliable the death was most likely caused by digoxin. The pediatric resident who cared for her until 5:00 p.m. on the day before death stated that her condition was stable and of no concern to him or to the nurses. Dr. Phillips, the Hospital Chief Pathologist, said that sufficient problems were found on autopsy to account for the baby's death. Dr. Bain agreed with Dr. Phillips' assessment and stated that the baby was at very high risk of death. Most of the other doctors conceded that if the toxicologic data were valid, digoxin at least contributed to her death. Drs. Hastreiter, Mirkin, Kauffman, and Fay have little doubt that upon the basis of the post-mortem serum digoxin level, digoxin was the main cause of death.

(34) CHARLON GARDNER

Diagnosis

This twenty-one day old baby girl suffered from a large ventricular and a small atrial septal defect. In addition, however, she had a most unusual and severe cardiac defect in that there was no pulmonary artery coming off the right ventricle. There was what is called a bilateral patent ductus arteriosus, that is, a ductus leading to each of the right and left lungs from the aorta, and it is only by means of these channels that blood can reach the lungs. In fact, it appeared that the ductus to the right lung was not functioning although some blood was reaching it through collateral vessels.

In the result, the blood coming to the right atrium and ventricle passed through the septal defect to the left ventricle and thence into the aorta. As there was no pulmonary artery, and consequently no shunt could be made from it to the aorta, everything depended on the ductus remaining patent which does not generally happen. There was a suspicion of Down's syndrome which was never confirmed.

Prognosis

The views of the cardiologists were as follows: Dr. Rowe - high risk death, Dr. Nadas - poor, Dr. Hastreiter - 9 (out of 10).

Course of Treatment

Upon admission on March 13, the child was cyanotic but feeding and breathing well. Since so much depended on the patency of the ductus, she was treated with prostaglandins to keep it open. Unfortunately, she suffered side effects of fever, persistent tachycardia and tachypnea from the drug, and the dose had to be reduced. Thereafter, she suffered from hypoxia, cyanosis when upset, an elevated temperature, and moderate heart failure. Digoxin was prescribed. On March 16 her heart rate was regular but her respirations continued to be shallow and laboured. She was very irritable and cyanosed. By March 17 she was alert and feeding well although she continued to have a fever and to be in moderate heart failure. Her heart rate was irregular as were her respirations.

(CHARLON GARDNER)

Terminal Events

At 2:30 a.m. on March 18 she became tachycardic and then experienced an episode of apnea. At 3:35 a.m. her apex became very irregular. The baby quickly went into ventricular fibrillation and at 3:45 a.m. a Code 25 was called but every effort to resuscitate failed. The terminal events included extreme bradycardia, gasping respirations, and atrioventricular block ("AV block" - dissociation of the beats between the two chambers). She was pronounced dead at 4:25 a.m.

Autopsy

The autopsy confirmed the heart defect and the patency of the left ductus and attributed death to her clinical condition. In the Final Autopsy Report the possibility of myocardial electrical instability and a fatal dysrhythmia were mentioned. Digoxin toxicity was not considered.

Toxicology

The baby was on digoxin but no ante-mortem or post-mortem blood levels were taken. Fixed heart and lung tissues were tested at the Centre of Forensic Sciences by RIA plus HPLC and RIA. The digoxin concentrations in heart tissue were found to be in the overlap area (within both the therapeutic and toxic ranges) and the levels in lung tissue were found to be exclusively in the toxic range. Once again, Mr. Cimbura estimated that the digoxin levels would have been greater in fresh heart tissue but he could not say how much greater.

The Experts

The Hospital doctors (Drs. Rowe and Freedom) were not concerned about this death at the time, attributing it to the heart disease and hypoxia. Drs. Hastreiter and Fay, and Dr. Cutz who conducted the autopsy, could not exclude digoxin toxicity but thought that as the cause of death it was unlikely. Dr. Nadas, in his report to Atlanta, rated the baby's death expected and consistent with her clinical condition, but also consistent with digoxin toxicity.

(35) ALLANA MILLER

Diagnosis

This baby girl, just under one year old at the time of death, had a complicated series of heart defects. There was no septum between the atria; the inferior vena cava instead of joining the right atrium joined the superior vena cava, and the veins from the liver drained directly into the right atrium. This venous abnormality usually interferes with the conduction system and patients are prone to have rhythm disturbances. She also had a condition known as polysplenia (the existence of multiple spleens). That condition is often associated with abnormalities of the lung.

The major cardiac defect was the common atria, which resulted in the mixing of blue and pink blood in the aorta and thence in the rest of the body, producing congestive heart failure. It is easier for the blood to go to the lungs than it is to be pumped through the aorta around the rest of the body, and the high pressure on the pulmonary artery produces blood vessel disease in the lungs.

Prognosis

The views of the cardiologists were as follows: Dr. Rowe - high risk death, Dr. Nadas - guarded, Dr. Hastreiter - 5 (out of 10).

Course of Treatment

The child had been in hospital in Kitchener (her native city) on several occasions and had been assessed and treated several times at the Hospital for Sick Children. Digoxin and diuretics had been prescribed for her congestive heart failure and a cardiac catheterization performed. She had failed to gain weight, and in early 1981, had again been admitted to hospital in Kitchener several times for chest infections, bronchiolitis (bronchopneumonia), and continuing heart failure. It was decided to admit her again to the Hospital for surgery in March, 1981, to close the large atrial septal defect. Dr. Freedom, the child's cardiologist, had hoped to defer surgery until she was older, but since her progress was unsatisfactory, he felt the surgery date had to be advanced. She was admitted to the Hospital at 10:37 p.m. on March 19. Her apex was irregular; she was congested, irritable and restless, pale and cyanosed, and throughout March 20 was still showing signs of heart failure, infection, and irregular heart rate, although sleeping and feeding better. Her heart monitor kept going off during the late evening. Her heart rate was unusually low for a child of this age, and she suffered from frequent arrhythmias.

Terminal Events

At 1:45 a.m. March 21 her heart rate became even more irregular and she had to be stimulated several times. She then gagged and vomited, and her respirations became very laboured. The resident was called and administered furosemide intravenously at 2:40 a.m.; at 2:45 a.m. the baby began to suffer seizures, developed extreme bradycardia, and went into cardiac arrest. Resuscitation efforts failed and death was pronounced at 3:27 a.m.

Autopsy

While the attending doctors were not surprised by the baby's death, they were already aware of the digoxin levels for Kevin Pacsai and the possible inquest into his death. Permission for an autopsy was granted, and the body was turned over to the Pathology Department. Dr. Costigan learned of the death at about 7:30 a.m. on March 21. He was suspicious of possible digoxin involvement in this death and concerned when he learned that no digoxin tests had been ordered. He attended at the pathology laboratory and asked Dr. Taylor to do a post-mortem digoxin test. Soon thereafter, Dr. Cutz independently asked Dr. Taylor to do the same thing. Late that afternoon Dr. Costigan, concerned that the digoxin tests might be postponed by the Biochemistry Department until Monday, March 23, spoke to Dr. Carver who expedited the testing procedure through Dr. Soldin. The results became known at 8:00 p.m. that day.

The Final Autopsy Report, while confirming the heart defects and disclosing evidence of heart failure and cardiac trauma associated with resuscitation, attributed death to digoxin toxicity because of the high post-mortem serum level.

Toxicology

Digoxin was held at 8:30 p.m. on March 19 until receipt of a level on March 20. The reading was .6 ng/ml (below the usual therapeutic range). One maintenance dosage of digoxin was ordered and administered at 9:00 p.m. on March 20 and a further "hold" order was made at 2:30 a.m. on March 21 just before her death. The result of the post-mortem test, conducted by RIA only, was initially greater than 50 ng/ml (approximately 72-73 ng/ml) but by March 22 was known to be 78 ng/ml. Dr. Soldin was satisfied that the tests had been performed correctly. He also arranged, on March 21, for a further sample of oral digoxin from the cardiac wards to be tested (as had been done after Kevin Pacsai's death) to determine if there was any error in the concentration reportedly contained in the preparation. His tests revealed no such error.

At 8:00 p.m. on March 21, Dr. Soldin reported the post-mortem serum results to Drs. MacLeod and Costigan. The latter immediately informed Dr. Carver who told Drs. Rowe and Fowler and instructed the latter to inform the Coroner. Dr. Carver then convened a meeting attended by Drs. Fowler, Costigan, and Mounstephen, and later by Dr. Tepperman, the Coroner. At this meeting, it was decided to make digoxin a "control drug" locked in the narcotics cabinet, to be dispensed only by the Team Leader or the Head Nurse, with the usual checks applied to controlled drugs which included signing by two nurses, and counting the supply at the beginning and end of each shift, and a memorandum was issued accordingly. Drs. Costigan and Mounstephen were dispatched to all wards to ensure this instruction was carried out and to check all crash carts for the drug and to remove it if found. These instructions the doctors carried out by the early hours of the morning on March 22; in fact, they found no digoxin on the crash carts on Wards 4A or 4B.

A post-mortem blood specimen was also tested at the Centre of Forensic Sciences using RIA only. A level of 69 ng/ml was detected. Fixed tissue samples of heart and lung were tested at the Centre of Forensic Sciences about three months after death and found to have only traces of pure digoxin in the heart; no traces were found in the lungs after testing by RIA and HPLC and RIA. A specimen of eye fluid was also tested using RIA only, and again only traces of digoxin-like substances were found.

Medication Error

Dr. Spielberg thought an accidental administration of digoxin instead of furosemide five minutes prior to the cardiac arrest might have occurred. This, he felt, could account for the very low levels of digoxin found in the fixed tissues since there would not have been sufficient time for distribution of digoxin to tissue. Dr. MacLeod agreed that this was possible. It was generally agreed by the experts that while some distribution might occur during the arrest, impaired circulation at this time would slow it down considerably. Dr. Kauffman, however, pointed out that in order to account for the post-mortem blood level by this hypothesis, it would take two errors: one to confuse furosemide for digoxin and another to give more digoxin than the volume of furosemide reported to have been given. Had the same volume of digoxin been given as had been ordered for the furosemide, the amount would in his opinion not be sufficient to account for the cardiac arrest of the child and her high post-mortem blood level. Dr. Hastreiter felt it unlikely that digoxin would have been confused for furosemide, and in any event, if it had, the concentrations of digoxin would not have reached those found in her post-mortem blood. Dr. Rowe, while he thought it was possible that an error had occurred, felt the accidental administration of digoxin was unlikely.

The Experts

The Hospital doctors (except Drs. Costigan and Cutz) were not suspicious of the death at the time it occurred. Dr. Kobayashi did however say that her death came as a surprise, and Dr. Freedom regarded it as unexpected. After hearing of the digoxin level, however, they all accepted digoxin toxicity as the cause of death. Dr. Bain stated that the infant's cardiac problems might well have caused her death. He added, however: ". . . the allegedly high levels of digoxin must be explained". Indeed, the

(ALLANA MILLER)

terminal events were consistent therewith; all of the outside experts agreed that digoxin toxicity was the main cause of death. Dr. Nadas, in his report to Atlanta, rated the baby's death unexpected and consistent with both her clinical condition and digoxin toxicity.

(36) JUSTIN COOK

Diagnosis

This three and one-half month old baby boy suffered from what is called "complex pulmonary stenosis". His heart was situated on the right side of his body instead of the left; the tricuspid valve which leads from the right atrium to the right ventricle was completely sealed off thus preventing the usual flow of blood to the right side of the heart. There was an atrial septal defect so the blue blood could pass through it, mix with the pink blood from the lungs, and pass through the mitral valve to what was essentially a single ventricle. From there, because of the stenosis of the pulmonary valve, there was difficulty in the blood getting to the lungs. Some would of course have passed through the ductus arteriosus but that would occur only so long as it was patent.

Prognosis

The views of the cardiologists were as follows: Dr. Rowe - high risk death, Dr. Nadas - poor, Dr. Hastreiter 8 (out of 10).

Course of Treatment

The baby was admitted to the Hospital in the late evening of March 20, 1981. He was catheterized the next morning and surgery (a Blalock-Taussig shunt) was scheduled for March 22. He did well following the catheterization, but at 6:00 p.m. suffered a near fatal blue spell, (i.e., the heart muscle started to contract intensely producing extreme cyanosis), and he had to be given the medication propranolol to reduce the contractions. He settled well after the blue spell but was placed on constant care nursing. He tolerated feedings at 8:30 p.m. and 2:30 a.m. and thereafter rested comfortably with regular heart rate and respirations.

Terminal Events

At 3:45 a.m., March 22, he appeared to be more cyanosed and began to have a seizure. A doctor was summoned and propranolol was given, but the baby remained cyanosed, and his respirations were laboured. Another dose of propranolol was given with partial response, but the baby's heart rate dipped, and atropine and morphine were administered. His heart rate continued to fluctuate, and at 4:20 a.m., when arrangements had already been made to transfer him to the I.C.U., he became bradycardic, developed ventricular fibrillation, and went into cardiac arrest. Resuscitation efforts failed and he was pronounced dead at 4:56 a.m.

Autopsy

The autopsy was limited (in the first instance) to the heart and lungs. The Final Autopsy Report noted the cardiac defects, mild to moderate pulmonary congestion and edema, and the post-mortem digoxin level; in consequence of the last, death was attributed to digoxin toxicity.

Toxicology

The child was not on digoxin. Indeed, the drug was contra-indicated because his particular cardiac condition made him especially susceptible to digoxin toxicity. By the time of the terminal events of this child, the doctors were aware of the elevated digoxin levels in Janice Estrella, Kevin Pacsai, and Allana Miller, and some of them had already attended meetings also attended by Coroners and Police. Dr. Jedeikin was present at the resuscitation and he took an ante-mortem sample of blood at about 4:30 a.m., and post-mortem blood samples, as well as samples of the fluid from the I.V. bag, between 5:00 and 6:00 a.m. The result on the ante-mortem blood specimen was 72 ng/ml and that on the post-mortem specimens varied between 68 ng/ml and greater than 100 ng/ml when tested at the Hospital by use of RIA, and between 46 ng/ml and 91 ng/ml when tested at the Centre of Forensic Sciences by use of RIA and HPLC and RIA. Mr. Cimbura arranged for a post-mortem specimen to be tested in a third laboratory at the Toronto General Hospital. The result was 100 ng/ml. The result of the I.V. fluid test performed at the Centre of Forensic Sciences was negative for digoxin. Fresh tissue samples of the heart, lungs, gastric contents, and small bowel (the latter obtained at a second autopsy) were also examined using RIA plus HPLC and RIA, and the levels were all found to be in the toxic range. The levels measured in the fresh heart tissue were almost the highest ever reported in toxic cases. There were very low levels of digoxin measured in fixed heart and lung tissues, examined three and one-half to five months later, again using RIA plus HPLC and RIA.

After the digoxin levels became known, Dr. Carver issued a second memorandum (see p. 149 ante for the first) to the effect that nursing supervisors would be assigned henceforth to Wards 4A/B to be responsible for the administration of all medications, and daily digoxin levels would be taken on all patients on those wards. All elective admissions to the wards were stopped; where possible, patients were transferred off the wards, and the nursing team for Ward 4A (of which Susan Nelles was a member) was to be relieved of duty.

Medication Error

There was much consideration, both in examination and in argument, of the possibility of digoxin dosage by error. Particularly, it was suggested that digoxin might have been given accidentally for propranolol shortly prior to the arrest. After the first blue spell at 6:00 p.m. on March 21, Dr. Kantak, on advice from Dr. Jedeikin, had ordered propranolol taped to the bedside for later use, and it was suggested that somehow digoxin had been mistakenly substituted. That does not seem possible. The drug was obtained by a nurse who testified that it was checked before delivery to her. Dr. Kantak, who gave both doses of propranolol, testified that he checked the vial to make sure it was correct before administering it. Certainly it was plainly labelled at the bedside.

There would have to have been multiple errors committed before digoxin could mistakenly have been administered for any of the drugs used at resuscitation. The first mistake would lie in its actually being on the crash cart. As mentioned earlier, Drs. Costigan and Mounstephen did not find any digoxin on the Ward 4A or 4B crash carts earlier in the night. There would have to have been at least one adult ampoule of digoxin on the cart (to account for the ante-mortem blood level); the physician who administered the drug, as well as the nurse who drew it up, would both have had to neglect to check the labelling of the ampoule before administration. Besides the propranolol doses discussed above, atropine was given at 4:00 a.m. and 4:10 a.m., morphine (which came from the medication cupboard) at 4:05 a.m., sodium bicarbonate at 4:23 a.m. (which comes from a much larger vial), and adrenaline at 4:29 a.m. It was argued that the blood sample used for the ante-mortem digoxin level might, in fact, have been taken later than 4:30 a.m. (the time listed on the biochemistry requisition slip made out for that sample). Adrenaline, sodium bicarbonate, and several other drugs were given intravenously between 4:32 a.m. and 4:55 a.m. Sufficient time must have elapsed, nevertheless, for distribution to tissue to take place in order to account for the very high tissue levels discussed under "Toxicology" above. Many of the same counter arguments apply to the submission relating to medication error at resuscitation as to the theory that digoxin was given inadvertently in place of propranolol. Drs. Spielberg, MacLeod, and Bain all said a medication error was possible. There was, however, no direct evidence of error whatever. Moreover, Dr. Kauffman testified that the theory of one adult ampoule of digoxin mistakenly administered during the resuscitation effort could only be valid if the post-mortem tests had shown little, if any, levels of the drug in the baby's tissues, that is, only if there had been no time for distribution. He also testified that digoxin administered in lieu of propranolol, in the same quantities intended for the propranolol, would not account for the levels measured post-mortem. Dr. Hastreiter as well, felt that administration of the drug in substitution for propranolol, or during the resuscitation, was unlikely. He felt the dose had been administered at or about 3:30 a.m. (at a time when no medications are recorded as having been given to the child).

The Experts

The terminal events are consistent with digoxin toxicity, but as is pointed out by Dr. Nadas in the Atlanta Report, the death although unexpected was consistent with the baby's clinical condition. He also expressed special concern regarding digoxin toxicity. In light of the digoxin levels obtained both before and after death, all doctors conceded that digoxin toxicity was a possible cause of death. Drs. Fay, Hastreiter, Mirkin, and Kauffman all found it the major precipitating cause. Dr. Rowe, upon learning of the child's digoxin levels, concluded that he had received an overdose of digoxin that caused his death. That remained his view when he testified before me. Dr. Bain, while conceding that the child received an unauthorized dose of digoxin, speculated that the infant's death might have been caused by a fatal blue spell which occurred prior to any toxic reaction caused by digoxin.

11. THE CONSIDERATIONS LEADING TO THE CONCLUSIONS

(a) The Categories

I am required by the Terms of Reference to report on how these thirty-six children came to their deaths. I think I should say at the outset that I can see only two realistic causes, and they apply to all of the children. The one cause is death from their cardiac (and in some cases extracardiac) conditions; the other is death from an overdose of digoxin either deliberately or accidentally administered; indeed, except in the cases of babies Velasquez, Hines, and Pacsai, no other cause was seriously suggested. I should also point out that if digoxin toxicity arising out of an overdose was the immediate cause of death it makes no difference under our law that the child's death was inevitable from his condition. Section 209 of the Criminal Code provides as follows:

> Where a person causes bodily injury to a human being that results in death, he causes the death of that human being notwithstanding that the effect of the bodily injury is only to accelerate his death from a disease or disorder arising from some other cause.

That is the test that I should use. If, on the other hand, the child died from his condition, but there is a possibility that therapeutic doses of digoxin adversely affected that condition and perhaps contributed to the death, that is, at least so far as the criminal law goes, a death from natural causes. Treatment in good faith and with adequate skill is not an offence. In any event I do not find that any of these children died from their prescribed treatment.

While subject to the reservations I have made, the cause of death may only be one or the other, that is, natural causes or digoxin toxicity, I, like the doctors, have often considerable difficulty in determining which of the two causes of death is appropriate. I have decided to assign the children into one of the following defined categories:

I. Deaths by Digoxin Toxicity (DT);

II. Deaths Highly Suspicious of Digoxin Toxicity (HS);

III. Deaths Suspicious of Digoxin Toxicity (S); or

IV. Deaths from Natural Causes (ND).

There may be a fifth category where I will be unable to designate the cause.

(b) Onus

There are two principles of law that might be thought to influence my decision as to the cause in any particular case; one is the fundamental principle of criminal law that the Crown must prove every necessary ingredient of the offence beyond a reasonable doubt. The other is the principle of civil law that where proof of a crime is an essential element in a civil cause of action, the more severe the consequences of the finding the more stringent should perhaps be the proof (see Smith v. Smith and Snedman, [1952] 2 SCR 312, per Cartwright J. at page 331). I do not consider myself affected by either of these principles. This is a Commission not a criminal proceeding, and although it resembles a civil proceeding, I am not permitted to identify any person who might have been involved in causing the deaths of any of the children; hence there can be no consequences for an individual so as to justify a more stringent standard. In my view, the appropriate standard of proof is the balance of probabilities applicable in a civil trial. By the very nature of the category system, however, I must necessarily be more certain of the matter when I place any death at one end or the other. Thus I will not attribute any death to digoxin toxicity or to natural causes unless I am fully satisfied it belongs there.

I think the point made by Mr. Lamek in argument is valid. One must be very reluctant to find that someone (even an unnamed person) has, in this world-renowned hospital, administered a fatal overdose of a drug to a baby under the Hospital's care. That obviously is one of the reasons for the lack of suspicion by the doctors and nurses throughout the epidemic period - even when the evidence was there for Janice Estrella - until the death of Kevin Pacsai. Nevertheless, once one is persuaded that one child has so perished, that reluctance is (or should be) weakened for any other child dying in similar circumstances. Once it is established for three or four, any reluctance for others is further weakened. Indeed, there may come a time when every death in similar circumstances might be approached with suspicion. I will not speculate where there is no evidence. Where there is evidence, however, I will not hesitate to consider it and reach a conclusion in accordance with the ordinary civil rule.

(c) Meaning of Categories

What then are the categories intended to mean?

I. "Digoxin toxicity" means that I have reached the conclusion that the child died of an overdose of digoxin. Generally speaking, I shall not be so persuaded in the absence of reliable toxicologic evidence so indicating, nor if other compelling evidence is inconsistent with that conclusion.

II. "Highly suspicious" means that I think the child died of a digoxin overdose. Generally speaking, these cases will be those where there is no reliable toxicologic evidence but the other evidence points strongly to digoxin toxicity as the cause of death.

III. "Suspicious" means that I do not know whether the child died of a digoxin overdose or from his clinical condition. These cases will be those where the terminal events are consistent with digoxin toxicity, and there is some other evidence pointing in that direction, but where there is also some evidence to support the theory of natural death.

IV. "Natural death" means that I have reached the conclusion that the child died from his clinical condition without the intervention of digoxin. These cases will be those where the terminal events are inconsistent with digoxin toxicity, or if they are consistent, where there is no other evidence, or insufficient evidence, to support that conclusion.

(d) Credibility of Witnesses

Where I have been faced with a conflict of evidence on some fact important to the issue, I have decided the matter in the usual way, generally by accepting the version of the witness whose evidence I found more convincing. Where it comes to the experts' views as to the cause of death, I must say now that I found the expert evidence of Dr. Hastreiter, the cardiologist, and Dr. Kauffman, the pharmacologist, where there was a conflict, preferable to that of the others in the same fields. They both were removed from the scene; they both approached the question without preconceived ideas, and they both had studied the problems thoroughly and gave their evidence in a consistent, credible, and persuasive manner. As to toxicology, I am satisfied as to the validity of Mr. Cimbura's methodology and results.

(e) Severity of Illness and Autopsy Findings

I should here mention two other factors which may have influenced the classification of each death. The first is the severity of the child's illness. By itself, it does not mean much. A very sick child may be deliberately (or accidentally) poisoned just as well as a healthy one. While he is more likely to die of his disease, some people, speculating on motive, might think the sicker the child, and perhaps even the more disfiguring or disabling the affliction, the more likely a deliberate poisoning might be. On the other hand, a child suffering from a relatively minor disease might be considered less likely to succumb to his condition, and perhaps, again for people speculating on motive, less likely to be deliberately poisoned. Notwithstanding the counter arguments that can always be made, I will point sometimes to the degree of severity of the illness when I think it in turn points to a particular conclusion.

The second matter relates to autopsy findings. I understand that it is not uncommon for an autopsy to fail to reveal the precise cause of death. Nevertheless, where it does fail to make such a precise finding and where, as here in some cases, there have been no toxicologic tests undertaken, and digoxin-related deaths have been found in others, one might reasonably wonder what a digoxin test undertaken at autopsy might have shown. The inability to establish a clear cause of death at autopsy may not be enough by itself to point to digoxin toxicity, but it does nothing to dampen the suspicion where it otherwise exists.

(f) Recommendations

While paragraph 3) of my Terms of Reference contemplates the possibility of recommendations, I do not think it appropriate to make any. The procedures at the Hospital were the subject of a very careful, detailed study in the Dubin Report and I cannot hope to improve on the recommendations there made. Moreover, our hearings did not extend specifically in either evidence or submissions to that subject; our concern was with the cause of death.

12. THE CONCLUSIONS ON PHASE I

I have endeavoured to place in Chapter 10 all the evidence that I consider relevant to a determination of the cause of death of each of the children. I shall now proceed, where I feel I can, to give an opinion on the cause of death of each particular child. In the course of that opinion, I may analyze some of that evidence. It would be wasteful to repeat all the evidence, but I must emphasize that it was a consideration of all the evidence that brought about my conclusions. In assessing the validity of those conclusions the reader is invited to return to Chapter 10, and indeed any other part of this Report that casts light upon facts set out in that Chapter.

I shall deal first with Category I, that is, "Deaths by Digoxin Toxicity", and I shall then proceed to Category IV which is "Natural Death". The reason is that by the very nature of those categories (and my definition of them) they are exclusions from the other two categories. It is much easier to be suspicious than it is to be sure, but it is much easier also to set forth your reasons when you are sure.

(a) Deaths by Digoxin Toxicity

The first child in this category was the last to die, namely, Justin Cook. It is difficult to think of any case where the evidence could be stronger. The child was not prescribed digoxin and highly toxic levels were found in his blood, both before and after death. The digoxin concentration measured in his fresh heart tissue (1177 ng/g) was almost the highest ever reported in a digoxin fatality case. The concentrations in his fresh lung tissue specimens were also in the fatal range. His death was unexpected, just before he was to undergo surgery, and his terminal events were consistent with digoxin toxicity. He was, of course, a very sick baby and might well not have survived long in any event, but the cause of death in my opinion was indubitably a massive overdose of digoxin.

The same I think can readily be said of Kristin Inwood and Allana Miller. While they were both prescribed digoxin and there were no toxic ante-mortem levels, they both had extremely high post-mortem levels (indeed, Kristin Inwood's serum digoxin level was the highest ever recorded and her fixed tissue levels were also extremely high). In each case the death was unexpected and while consistent with the child's clinical condition was also consistent with digoxin toxicity. While each could have died of her clinical condition, the toxicologic evidence persuades me, as it did Drs. Hastreiter, Mirkin, and Kauffman, that these children died of overdoses of digoxin.

Next in this category is Kevin Pacsai. While he had a lower post-mortem digoxin level than the three children discussed above (except at Mount Sinai Hospital), he had an ante-mortem level which in itself would excite suspicion. He was also a much healthier child. Like Drs. Kauffman and Hastreiter I cannot accept the theory of transient adrenal insufficiency; like Dr. Kauffman I cannot attribute the high digoxin level to some pathophysiologic cause. This was a child who should not have died and the only rational explanation for his death is digoxin overdose.

There are three further babies who, in my opinion, belong in this category; they are Jordan Hines, Stephanie Lombardo, and Jesse Belanger. None was prescribed digoxin, and all were found to have it in their tissues. With Jordan Hines there were both fixed and exhumed tissues, with the others only exhumed. As I have said, digoxin concentrations in exhumed tissues mean nothing quantitatively and in fixed tissues little. But the one overwhelming fact is that the digoxin was there at all.

The fact that a baby's test discloses digoxin where none was prescribed does not by itself determine conclusively that the baby died of the drug, but the overpowering inference in these circumstances is that all three did. I have indicated in the individual summaries the improbability of mistaken administration. I would again emphasize how unlikely it is that there could be accidental and unreported administration of unprescribed digoxin to three babies (four including Justin Cook in whose case I have rejected the possibility of digoxin error, see ante at pages 154-155) on the same wards within so short a space of time. Assuming there was no error, the administration could only be for the purpose of causing harm to the baby. That the administration did cause harm (indeed the death of the baby) is almost the inevitable conclusion.

I do not accept the diagnosis of sudden infant death syndrome for Jordan Hines. As I noted earlier, Dr. Kauffman found the child's course in the Hospital quite inconsistent with it. It may or may not be that one or more of the episodes of apnea he suffered earlier might have led to death if there had not been prompt intervention, but the chance of a sudden infant death when the child had two monitors attached and was under regular nursing care, while not unknown, cannot be very great. As Dr. deSa said, SIDS is a diagnosis by exclusion, and one cannot exclude digoxin toxicity as a cause of death. The child might have been suffering from sick sinus syndrome or some other defect of the conduction system, but I accept the opinion of Dr. Hastreiter that death therefrom was unlikely.

We will never be sure, of course, whether the hypothesis of the occluded shunt was valid for Stephanie Lombardo, but it is a theory for which there is no physical evidence. For the theory of digoxin toxicity there is hard evidence. I have no difficulty in favouring the latter.

With Jesse Belanger the toxicologic evidence is not quite so strong, but his Blalock-Taussig shunt was found intact at autopsy. His death was unexpected and (according to Dr. Nadas) inconsistent with his clinical condition. In light of the unprescribed digoxin, I accept the opinion of the outside experts that digoxin toxicity is the probable cause of death.

There is just one other child who comes within this category and that is Janice Estrella. Her position is exactly the same as that of Kristin Inwood and Allana Miller, except in her case there is considerably more ground for suspecting the integrity of the post-mortem digoxin level. As I have stated, that reading is now under heavy attack because of possible contamination and because of the one example of a grossly elevated level in the study conducted by Mr. Cimbura and Dr. Phillips (discussed under this baby in Chapter 10). I am disinclined to disregard the reading as did so many of the experts. Like Dr. Mirkin I tend to the view that 25 out of 26 (2 of the 28 samples were not tested) is more confirmatory than 1 out of 26 is contradictory.

I am suspicious of the January 7 incident (which also took place in the early morning on the same ward with the same nursing team in attendance) shortly after which a digoxin level was measured as greater than 9.4 ng/ml. That is a very high level and although I recognize that it could be explained by renal or pre-renal failure, I do entertain suspicion that the level may have been produced by the administration of an unprescribed dose of digoxin. The post-mortem level of greater than 4.7 ng/ml in the leg vein sample, although again perhaps explainable by the post-mortem multiplier factor, does not dispel my suspicion. And if my suspicion is reasonable, it becomes even easier to infer that her death was digoxin-related.

This was a sick baby, I will concede. But her surgery had been performed and the repair was found intact at autopsy. The death was unexpected, and an overdose of digoxin seems to me the most likely cause.

(b) Natural Deaths

I proceed now to the other end of the scale: those babies who died natural deaths. Foremost among these are Alan Perreault, Paul Murphy, Lauretta Heyworth, Bruce Floryn, and David Leith. I need only say a few words about these poor afflicted children. According to Dr. Nadas, the course of terminal events of the first four of these children was inconsistent with digoxin toxicity. For Alan Perreault his unhappy life lasted longer than expected and there was no sign of digoxin in what might have been a post-mortem blood sample. Paul Murphy was a fourteen year old boy who was dying on his last admission. The death itself was sudden but manifested none of the other symptoms of digoxin toxicity. The eleven year old Lauretta Heyworth was also in an extreme condition on admittance and her condition had steadily deteriorated. The suddenness of her terminal events induced Dr. Hastreiter to believe there was a small probability of digoxin toxicity, but there is nothing else to place her outside the "natural death" category, and I would decline to do so.

Bruce Floryn's death created no controversy among the experts. There was nothing to raise suspicion. The same applied to the baby David Leith, although the non-testifying expert (Dr. Nadas) found the death consistent with digoxin toxicity. I would unhesitatingly place both these children in the "natural death" category.

Another baby who belongs in this category is Francis

Volk whose terminal events in the opinion of all experts were not consistent with digoxin toxicity. He was a very, very sick baby whose condition brought about his death.

(c) Highly Suspicious Deaths

I will move on to those whose deaths I consider highly suspicious, that is, those who I believe died of an overdose of digoxin but for whom there exist no reliable toxicologic data to support the belief.

The first of these is the first baby to die in our amended time frame, that is, Laura Woodcock. Her death was unexpected; indeed, she had a good prognosis and should not have died. Her death was also not explained by the Final Autopsy Report. Dr. deSa, it will be remembered, regarded her case as one of three not adequately explained by the pathologic evidence. She was not on the Tour End Report, and she had been stable for four days. The only other suggested causes of death, namely her liver disease and pneumonia, were ruled out by Drs. Hastreiter and Mirkin. She had only a minor cardiac problem and a good prognosis and should not have died when she did.

The second baby is Amber Dawson. She too had a good prognosis and her death was considered by most of the experts to be quite unexpected. At autopsy her surgical repair was found intact and the pathologic findings did not explain her death. Thus, neither her clinical condition and course nor the autopsy provide any convincing explanation for her having died when or in the manner she did. It is, of course, most unfortunate that the opportunity to determine the matter by a post-mortem digoxin test was not taken. On the basis of the evidence there is, I believe the death likely to have been brought about by a digoxin overdose.

The third baby is Antonio Velasquez who was recovering from surgery when he suffered his arrest and death - an event that was not only unexpected and inconsistent with his clinical condition but consistent with digoxin toxicity. No one offered any explanation except the exceedingly rare (almost unknown) idiosyncratic reaction to naloxone. I think all the doctors who at the time accepted that cause would not have done so if any other reasonable cause was available. I think there is another cause, namely digoxin toxicity, and it is to that that I believe this death should be attributed.

I must confess that I reached the conclusion almost by the process of elimination. The death was not attributed to codeine; it was attributed to naloxone, but that attribution I cannot accept. What then could have been the cause of death in a relatively healthy baby? I appreciate that there can be unexplained natural deaths, but in these circumstances, with my conviction that many babies on these wards, at this time of night, in the presence of members of the same nursing team, met their deaths by reason of massive overdoses of digoxin, I must be very suspicious that the same fate befell him.

The next baby in this category is John Onofre. While the autopsy disclosed many potential causes of death, I think the main important finding is that his Blalock-Taussig shunt was still intact. Perhaps it was too small to sustain life, but obviously that had not been the view of the surgeon. I accept the conclusions of Dr. Nadas and Dr. Rowe as to the unexpectedness of the death and of Dr. Nadas as to the existence of special concern with respect to digoxin toxicity. I also accept Dr. Hastreiter's opinion that the probable cause of death was digoxin overdose.

The last baby in this category is Real Gosselin. This baby came from Winnipeg and died the day after arrival at the Hospital. The death was totally unexpected. The surgery originally planned for the day he died had been postponed one day, no doubt because of worry about the digoxin level, but also without any expectation of intervening death. The only explanation of that death is the alleged failure of the prostaglandin treatment to keep the ductus open. It is clear that the treatment was not entirely working, but the child was stable before his terminal events and the ductus is noted as "patent" in the Final Autopsy Report. While a baby in his condition certainly can die suddenly without warning, I am deeply suspicious that this baby died not from his condition but from a digoxin overdose.

(d) Suspicious Deaths

There are certainly suspicious circumstances in the deaths of other babies. The deaths and terminal events of all of the following children were considered in the Atlanta Report to be consistent with digoxin toxicity. In addition, I have outlined some of the other suspicious circumstances. These circumstances are not however, in my opinion, sufficient to justify the conclusion or even the belief that the babies died from digoxin toxicity. Equally, in light of these circumstances, I cannot classify the deaths as "natural".

David Taylor's death was in all probability inevitable in the near future. His terminal events were markedly symptomatic of digoxin toxicity and they concerned Dr. Izukawa long before there was any general concern about digoxin. There is also the indication by Dr. Mirkin that the electrocardiogram reading suggested digoxin toxicity.

Philip Turner too was extremely sick and headed for almost inevitable death; nevertheless, the operation had been a success, and post-operatively he had been improving. One must consider the possibility, as did Dr. Hastreiter, that a digoxin overdose brought about this unexpected (as to time) death.

Dion Shrum in his terminal events manifested classic symptoms of digoxin intoxication. His death was unexpected and Dr. Hastreiter thought there was at least a fair probability of digoxin toxicity.

Brian Gage's condition was certainly not hopeless, yet he died early on the morning of contemplated surgery. The autopsy failed to find a definitive cause of death. The death was unexpected and one cannot exclude digoxin toxicity as the cause. Dr. Kauffman was sufficiently concerned about the terminal events to give the child a digoxin score of 2.

Richard McKeil had undergone surgery and both his shunt and the banding were found intact at autopsy. The main concern of his treatment was to improve his nourishment. Nothing on the night before could have presaged the sudden onset of terminal events.

There was really little reason to expect Darcy MacDonald's sudden demise when successful corrective surgery was contemplated. I am concerned about the suspicions of the resident at the time of death and the failure of the autopsy to show a precise cause of death. I am also mindful of Dr. Hastreiter's opinion regarding the cause of this child's death.

Jennifer Thomas, notwithstanding her fatal disease, was stable and feeding eagerly on her last night. She died suddenly in the early morning of the day scheduled for her operation. At autopsy, her ductus was patent. The toxic digoxin level in her fixed lung tissue, while not in itself conclusive, may heighten one's suspicion of digoxin toxicity.

Colleen Warner was one of the sickest babies during the period, but nevertheless, her death very shortly after admittance to the ward was both sudden and unexpected. The toxicology (fixed tissue) is certainly not conclusive but does nothing to allay one's suspicion.

I consider Barbara Gionas' death suspicious largely because she had improved considerably and the resident who attended her until just before her death was surprised by it. Both Drs. Kauffman and Hastreiter were prepared to consider digoxin toxicity seriously as a cause of death, and Dr. Nadas found the death consistent with special concern for digoxin. I also do not think I can ignore Dr. Kauffman's digoxin score of 2, although I am not entirely happy with his rating scheme.

Charlon Gardner seemed to be reasonably stable the day before death. Although she had trouble with the prostaglandin treatment, her left ductus was still open on autopsy. While the toxic fixed lung tissue readings are themselves insufficient, they do nothing to dispel suspicion.

(e) Undesignated Deaths (UD)

This leaves the following children outside the categories I have set forth: Andrew Bilodeau, Lillian Hoos, Kelly Ann Monteith, Antonio Adamo, Matthew Lutes, Frank Fazio, and Michelle Manojlovich.

I have examined and re-examined the evidence with regard to these babies and I must now confess that I simply cannot reach a conclusion. The choice of category is between "Natural" death and "Suspicious" and I considered for a while creating a separate category of "low suspicion" but eventually decided against it. In each case there are features that prevent my being satisfied that the death was attributable to natural causes, but those features are not so strong as to justify my placing the death in the "Suspicious" category. There is not in my view sufficient information to enable me to make an intelligent judgment.

(f) Summary

In the result, therefore, I would place the deaths of these children in the following categories -

I. Deaths by Digoxin Toxicity:

Stephanie Lombardo
Jesse Belanger
Janice Estrella
Jordan Hines
Kevin Pacsai
Kristin Inwood
Allana Miller
Justin Cook

II. Deaths Highly Suspicious of Digoxin Toxicity:

Laura Woodcock
Amber Dawson
Antonio Velasquez
John Onofre
Real Gosselin

III. Deaths Suspicious of Digoxin Toxicity:

David Taylor
Philip Turner
Dion Shrum
Brian Gage
Richard McKeil
Darcy MacDonald
Jennifer Thomas
Colleen Warner
Barbara Gionas
Charlon Gardner

IV. Deaths from Natural Causes:

Alan Perreault
Paul Murphy
Lauretta Heyworth
Francis Volk
Bruce Floryn
David Leith

V. Deaths Undesignated as to Cause:

Andrew Bilodeau
Lillian Hoos
Kelly Ann Monteith
Antonio Adamo
Matthew Lutes
Frank Fazio
Michelle Manojlovich

(g) Alphabetical List of Deaths:

Concordance with Chapter X

Adamo, Antonio (UD)	84
Belanger, Jesse (DT)	102
Bilodeau, Andrew (UD)	54
Cook, Justin (DT)	152
Dawson, Amber (HS)	59
Estrella, Janice (DT)	105
Fazio, Frank (UD)	111
Floryn, Bruce (ND)	113
Gage, Brian (S)	79
Gardner, Charlon (S)	145
Gionas, Barbara (S)	127
Gosselin, Real (HS)	95
Heyworth, Lauretta (ND)	77
Hines, Jordan (DT)	121
Hoos, Lillian (UD)	63
Inwood, Kristin (DT)	141
Leith, David (ND)	117
Lombardo, Stephanie (DT)	98
Lutes, Matthew (UD)	88
MacDonald, Darcy (S)	93
Manojlovich, Michelle (UD)	130
McKeil, Richard (S)	81
Miller, Allana (DT)	147
Monteith, Kelly Ann (UD)	70
Murphy, Paul (ND)	72
Onofre, John (HS)	90
Pacsai, Kevin (DT)	133
Perreault, Alan (ND)	52
Shrum, Dion (S)	67
Taylor, David (S)	56
Thomas, Jennifer (S)	115

Turner, Philip (S)	65
Velasquez, Antonio (HS)	74
Volk, Francis (ND)	86
Warner, Colleen (S)	119
Woodcock, Laura (HS)	49

On the evidence I cannot find that any one of the deaths that I conclude or believe or suspect were caused by digoxin toxicity was the result of accident or medication error.

C. THE INVESTIGATION AND PROSECUTION - PHASE II

13. THE FACTS UP TO THE ARREST

(a) The Coroners

As we have seen, Dr. Fowler reported the death of Kevin Pacsai to the Coroner on March 12, 1981, the day of the baby's death. The coroner designated was Dr. Paul Tepperman and he, in accordance with routine, ordered an autopsy to be performed at the Hospital under a Coroner's Warrant. As we have also seen, Dr. Costigan obtained an ante-mortem sample of the baby's blood and gave it to the biochemist for testing, and Dr. Cutz, who conducted the autopsy, ordered a post-mortem digoxin test.

On March 16, according to Dr. Tepperman, Dr. Fowler telephoned him to report that the post-mortem digoxin blood test had produced a reading of 25-26 ng/ml. (According to Drs. Carver, Rowe, and Fowler, the date of this call may have been March 17 or March 18.) Sometime, probably on March 20 (Friday), Dr. Cutz told Dr. Mancer of the Pacsai digoxin reading, and as Dr. Mancer put it: "Suddenly the Estrella case with its level of 72 ng/ml assumed new importance to me". Dr. Mancer then did two things: first, he went to see Dr. Ellis, the biochemist, and discussed with him the validity of the Estrella reading - he learned that it had been tested through several assays on dilution which made the possibility of mathematical error most unlikely - and secondly, he called Dr. Tepperman and informed him that not one but two babies had died with elevated digoxin levels. Dr. Tepperman immediately proceeded to Dr. Ellis' office, discussed the digoxin levels with him, and read the Estrella chart. At that time he also learned of Kevin Pacsai's ante-mortem digoxin level.

The next day, Saturday, March 21, Dr. Tepperman notified the Acting Chief Coroner, Dr. Ross Bennett, of the developments, particularly the two elevated digoxin levels in babies at the Hospital; Dr. Bennett called in two homicide detectives, Staff Sergeant Jack Press and Sergeant Anthony Warr, to help in the investigation. The Police were, however, at that point brought in to assist the Coroners; there was not at that time any sense that it was a Homicide investigation. Dr. Bennett then convened a meeting of Hospital personnel, Police, and Coroners.

The meeting took place at Dr. Bennett's office at 1:30 p.m. Drs. Rowe, Fowler, and Carver, and administrative officers were present from the Hospital; the two above-mentioned Police officers attended also as did Drs. Bennett, Peter King (the Regional Coroner), and Tepperman from the Coroner's office. The high digoxin levels were discussed. Dr. Rowe made mention of a "six-month study" of patient deaths (presumably he was referring to the meetings described in Chapter 4 supra) and that a further study was to start the following Monday. (Dr. Rowe had testified earlier that he had no recollection of mentioning a further study or indeed of any further study being in prospect). It was agreed the Police investigation would start the following Monday, March 23. There was some discussion about the possibility of further misuses of digoxin. The Hospital staff seemed to think they had the drug under control (as we have seen, at that time the only action had been to check the concentration of the oral preparation and the dosage given to Kevin Pacsai; see page 136 ante). To the shock of the Hospital doctors, the Police suggested the possibility of murder. Dr. Tepperman testified that he left the meeting thinking an innocent explanation would eventually be forthcoming. The one thing all agreed on was the need for absolute secrecy of the investigation to avoid panic in parents of patients or potential patients of the Hospital. As we know, by this time Allana Miller had died, and a post-mortem digoxin test had been ordered. The reading had not yet been reported, and her death was not revealed at the meeting.

When that level became known at about 8:00 p.m. and Dr. Carver was informed, he took the action previously referred to (see ante page 149); he also instructed Dr. Fowler to inform Dr. Tepperman. This he did, and Dr. Tepperman later joined the meeting then in progress. Dr. Carver asked Dr. Tepperman if the nursing team should be withdrawn. It was the first time the latter appreciated that one team was a common thread but, still suspecting nothing sinister, he said he did not think it could be justified. Dr. Tepperman telephoned Dr. Bennett to report Allana Miller's death and post-mortem digoxin level; together they decided the investigation should commence the next morning, and Dr. Bennett advised the Police accordingly.

At 5:30 a.m., Sunday, March 22, Dr. Fowler advised Dr. Tepperman of Justin Cook's death. Dr. Tepperman informed Staff Sergeant Press, but there was no particular alarm because the digoxin levels were not yet known. Since Justin Cook was not receiving digoxin, and since he was gravely ill, his death was not yet necessarily considered suspicious. From the time Justin Cook's digoxin levels were known it became essentially a police investigation.

(b) The Police

As I have said, Staff Sergeant Press and Sergeant Warr were the officers detailed to assist the Coroner. In the course of the investigation, they were assisted by, among others, Staff Sergeant Donald Sangster, Sergeant John Barbour, and Police Constables Stephen Hulcoop and John Murray, and were advised from time to time by their superiors Chief John Ackroyd and Deputy Chiefs Jack Marks and Thomas Cooke. It is not the practice of the Metropolitan Toronto Police Force to place a higher ranked officer in active charge of any investigation. Reports are made daily and the senior officers are kept informed. They control the investigation in the sense that they control the investigators, but they rarely participate in it.

Staff Sergeant Press and Sergeant Warr prepared a plan of operation for the investigation after the Saturday meeting. In all, the officers prepared themselves with twenty-eight specific questions. They attended at the Hospital at about 10:00 a.m. on March 22 and met with Drs. Carver, Rowe, and Fowler as well as other physicians and representatives from nursing and the Hospital Administration throughout the course of the day. They requested the medical charts of the four children (babies Estrella, Pacsai, Miller, and Cook) and met with Dr. Costigan and others to review them. Staff Sergeant Press may have then requested the records of all the patients who had died on the wards since January 1, 1981; he did request the records of the staff on the wards, details as to the manner in which digoxin was administered, the records kept of its use, and certain Pathology reports.

At about 2:00 p.m. the results of Justin Cook's digoxin tests became known. (For some reason Dr. Tepperman was not informed until 11:00 p.m.) The Police officers also became aware that the same nursing team had been on duty for the deaths of each of the four children and that they were scheduled to return to duty that night. They, together with the Hospital Administration, decided that the team should be relieved of duty that night and that decision was carried out. The team would, in the ordinary course, next report for duty commencing at 7:15 a.m., Wednesday, March 25.

By the end of the day, the Police were aware of the following:

(i) there were four babies who were known to have died with elevated digoxin levels, namely, Janice Estrella, Kevin Pacsai, Allana Miller, and Justin Cook;

(ii) Justin Cook was on constant nursing care on the night of his death, the nurse being Susan Nelles;

(iii) one nursing team had been on duty for all four deaths;

(iv) there was a consensus of opinion that the four children had died of an overdose of digoxin;

(v) the Hospital doctors agreed that the most likely method of digoxin administration was by intravenous injection, and the death or the terminal symptoms of death would have occurred at "the end of the needle", or almost instantaneously; and

(vi) the janitorial staff, cardiac fellows, and residents were not generally on the wards after midnight. A fellow and some residents were on call, the latter actually sleeping in the Hospital.

During that day also, Sergeant Warr attended with Dr. Fowler to survey Wards 4A/B. He reported back to Staff Sergeant Press that while in Dr. Fowler's words the wards were "mirror images of each other" they were separate independent entities with separate nursing teams and separate medication rooms. Both Police officers assumed there was little passage from one to the other, an impression that stayed at least with Staff Sergeant Press for many months.

The following morning, Monday, March 23, Staff Sergeant Press and Sergeant Warr reported to their superiors. The former had spoken directly to Deputy Chief Marks the day before, reporting the four deaths and his suspicion of foul play, and that morning told Chief Ackroyd that he was reasonably satisfied there had been deliberate killings at the Hospital. He proceeded to set up an investigative office at the Hospital and instructed Staff Sergeant Sangster and Sergeant Barbour on their duties. At 10:00 a.m. he attended another meeting of Police, Dr. Bennett, Hospital doctors, and Hospital administrative staff, at which there was discussion of the deaths on the wards since July, 1980. The Hospital agreed to provide the charts of all such patients for Police examination. Everyone was still most anxious to keep the investigation secret for the time being. There was further discussion of the timing of the overdose, and the specialists modified the "end of the needle" concept to a maximum time from dosage to onset of critical symptoms of fifteen minutes.

At that meeting there was some discussion of the possibility of oral administration and to rule out that possibility it was decided to examine Justin Cook's gastric contents. Accordingly, Dr. Bennett arranged for a further and more complete autopsy of that baby in Owen Sound, where the body had been returned to the family for burial, and for the delivery of the specimens obtained to the Hospital for digoxin testing. The Hospital did assays on these specimens and Dr. Ellis telephoned Staff Sergeant Press late on the evening of March 24 to report the preliminary results. He told him that he was unsure of their meaning. As we have seen (ante page 23), the Hospital decided on March 25 against doing more assays on tissue and these specimens were later taken to the Centre of Forensic Sciences for further testing. The Police were also provided with and examined the nursing assignment books for Ward 4A for December 23, 1980 to March 21, 1981, and for Ward 4B for January 8, 1981 to March 17, 1981, and the most recent Ward Information Nursing Statistics' (WINS) sheets. From these, it was apparent that Susan Nelles was charged with the care of Kevin Pacsai, Allana Miller, and Justin Cook, the last under constant nursing care, on the shift on which their deaths occurred. It was also apparent that she was not on duty at the time of Janice Estrella's death.

The evidence of deliberate overdoses for all four babies was strong, for Justin Cook overwhelming. The Police appreciated the situation as follows:

(i) that constant nursing care meant what it said - the nurse did not leave the care of that child to anyone else. They knew, however, at the latest by

Tuesday, March 24, that a nurse on constant nursing care was relieved for breaks, some short, some as long as one hour, by another registered nurse, usually the Team Leader, who took over at the bedside;

(ii) that the maximum time from overdosage to onset of critical symptoms was fifteen minutes. This was indeed the evidence until the Preliminary Inquiry, and even there Dr. Hastreiter, while expanding the possible time to thirty minutes, maintained the likely critical period to be fifteen minutes, at least for Justin Cook. Because digoxin was contraindicated for him, the baby would be more readily affected by it. The onset of critical symptoms according to his chart was 3:45 a.m. making the beginning of the critical period 3:30 a.m. All the evidence then available to the Police placed Susan Nelles alone with Justin Cook during that critical period;

(iii) that Susan Nelles had both Allana Miller and Kevin Pacsai under her care. The investigators particularly noted that Kevin Pacsai had suffered the onset of critical symptoms on Ward 4B to which Susan Nelles was assigned on that particular night. They noted that she had charted the terminal events for both babies, including the period just before the onset of critical symptoms. They assumed that meant that she was caring for the child during the period described. In fact, the nurse assigned to the patient regularly charts all developments, even those taking place when she is relieved. Also contrary to the Police belief at the time, nurses did not make a note in the patient's chart concerning the timing of relief breaks or by whom they were relieved; and

(iv) that while Janice Estrella was not in the care of Susan Nelles at the time of the onset of her critical symptoms - 2:40 a.m., January 11 - Susan Nelles had been on duty until 7:45 p.m. on January 10. Sergeant Warr (who had some personal familiarity with I.V. lines) noted from the baby's chart that her I.V. line had gone interstitial (i.e., the line had come out of the vein and the fluid was

> passing into the surrounding tissue) at 4:45 p.m., that it had been restarted at 6:00 p.m., but had gone interstitial again. He considered the possibility of slower infusion of the drug taking several hours to reach its fatal effect. He discussed the theory, although not in detail, with Dr. Freedom who viewed it as a "viable theory". He also discussed it later with Drs. Bennett and Tepperman who considered it possible. In fact, the I.V. line was again correctly situated in the vein by 7:30 p.m. (when an I.V. medication was administered), but this fact was not recorded in the baby's chart.

The Police, not unreasonably, discounted any theory of there being more than one killer. The suspicion of Susan Nelles as the killer of all four babies hardened.

The Police, possibly with the assistance of Drs. Costigan and Tepperman, prepared a large Bristol-board chart of twelve of the babies who died between January and March, 1981; these babies were Estrella, Fazio, Thomas, Warner, Hines, Gionas, Pacsai, Manojlovich, Inwood, Gardner, Miller, and Cook. Dr. Tepperman, after reviewing the medical records, decided that the deaths and terminal events of ten of the twelve babies were similar and these ten were: Estrella, Fazio, Warner, Gionas, Pacsai, Manojlovich, Inwood, Gardner, Miller, and Cook; he rejected the babies Hines and Thomas. (According to Staff Sergeant Press, he also rejected Charlon Gardner.) Initially, the chart set forth the particulars of the children and the digoxin levels where applicable. Later, information was added concerning the nurses on duty at the relevant times, and other pertinent data.

On Tuesday, March 24, the investigation of course continued. There was a further meeting at 10:00 a.m. of the Police, Dr. Bennett, doctors, senior nurses, and administrators from the Hospital. The four deaths where toxicologic evidence was present (babies Estrella, Pacsai, Miller, and Cook) were discussed. There was renewed advice from the doctors that the time from administration of digoxin to onset of critical symptoms was at most fifteen minutes. There was discussion of the duties and work schedules of cardiac fellows, residents, and nurses, and there was further consideration of the matter of security in keeping the investigation, for the time being, from becoming known to the media.

In the afternoon a meeting was held at the Hospital at which the Police (Staff Sergeants Press and Sangster, Sergeants Warr and Barbour) and Drs. Bennett and Tepperman were present. In addition, however, Mr. Jerome Wiley, assistant Crown Attorney of the County of York, was present by special invitation. Staff Sergeant Press explained the course of the investigation and the Police suspicion, with the reasons therefor, of Miss Nelles. He also said it was the intention of the Police to interview her, as well as the other members of her nursing team the following day. It was their intention also, if nothing came out of those interviews, to arrest Susan Nelles on the charge of the murder of Justin Cook. Mr. Wiley was most uncomfortable and initially found it hard to believe that Justin Cook's death was caused by a digoxin overdose. Dr. Tepperman explained all the medical facts of the case to him and gave his opinion that digoxin had caused that death. Finally, when pressed as to whether an arrest should be made, Mr. Wiley expressed his satisfaction that there were reasonable and probable grounds (a reference to section 450 of the Criminal Code which allows a police officer to arrest without warrant where those grounds exist). Indeed, he may have used the colloquial expression (as reported and perhaps translated by Staff Sergeant Press): "You have reasonable and probable grounds coming out your ears". Drs. Bennett and Tepperman expressed no opinion on the propriety of the contemplated arrest. They perhaps were taken by their silence to approve, but I think that they considered the question not their affair. It was now clearly a police investigation and they were present only to assist.

Since Miss Nelles was under heavy suspicion it was thought advisable that she be cautioned before any question was put to her, and an adaptation of the usual police caution was devised, and approved by Mr. Wiley. It was the usual police caution less the opening line indicating a charge was being made. The precise form was: "You are not obliged to say anything unless you wish to do so but whatever you say will be taken down and may be given in evidence".

Following that meeting, Dr. Bennett and the Police had a further meeting with Dr. Rowe and Messrs. J. Douglas Snedden and Kenneth Rowe of the Hospital Administration to inform them of the progress of the investigation, the intention of the Police to interview the nurses, and the concentration of their suspicions upon Miss Nelles. It is possible that at the meeting or shortly thereafter some member of the Hospital staff, either Dr. Fowler or Mr. Kenneth Rowe, told Dr. Bennett that they (i.e., the senior staff of the Hospital) also suspected Miss Nelles.

Mr. Wiley, on leaving the earlier meeting, informed his superior John Takach, Q.C., the then Director of Crown Attorneys, of the investigation into the deaths at the Hospital. They discussed particularly the case of Justin Cook, although Mr. Wiley did express the concern that many more babies' deaths were involved. They considered the possibility of accident, but finally Mr. Takach concurred (despite some reservations he had about possible medication error and the timing and institution of the proceedings) that there were reasonable and probable grounds for the contemplated arrest of Susan Nelles for the murder of Justin Cook.

There are two other matters that should be discussed before we leave the events of March 24. First, acting on some information received from Dr. Fowler via Dr. Bennett, Sergeant Barbour interviewed the former and was informed by him that he attended the Hospital shortly after the death of Justin Cook and in Dr. Fowler's words: ". . . [Susan Nelles] had a strange look on her face that was not in keeping with a nurse who had just had a person die that was under her care". It is not evidence that impresses me; nor did it impress Judge Vanek. Nevertheless, the Police found it confirmatory of their suspicions and the Crown tendered it at the Preliminary Inquiry.

The second matter is the problem of the nursing team's return to the Hospital to perform their duties. As I have said, they were relieved of duty on March 22 until the morning of Wednesday, March 25. There seems to have been some impression, at least on the part of Dr. Bennett and Mr. Wiley, that the Hospital was pressing to return them to duty on that day. I am quite satisfied that this was not so. The Hospital agreed to keep them off duty until the Police were satisfied they could return. Indeed, the Hospital on March 24 did instruct the Head Nurse to inform each member of that team to remain off duty until further notice, and the team was so advised. The Police felt no pressure whatever to take any unusual action to prevent the return of the nurses, nor to make an early arrest to prevent that return.

I pause now, to make certain reflections. It is the duty of the police to investigate, and where appropriate arrest, all of course in accordance with law. The police are not the servants either of the Crown Attorneys or of the Coroners. A police officer may (as here) sometimes be provided to the Coroners to assist them in a Coroner's investigation or inquest, and police officers in contemplating an arrest sometimes (as here) seek the advice of Crown Attorneys as to whether or not a charge should be laid. But once it becomes a Homicide investigation (as it did here with the knowledge of Justin Cook's digoxin levels), the police act independently of the Coroners, and regardless of the advice of Mr. Wiley, the discretion to arrest and charge lay with the Police, in this case with Staff Sergeant Press who was in charge of the investigation.

The second observation I make is that I was very much impressed with the thoroughness, the scope, and the speed of the Police investigation in the first few days. I have noted many false impressions that the Police gathered during that period, but it must be remembered that this was a field entirely foreign to them and filled with technical and scientific matters beyond a layman's ordinary comprehension. By the end of the third day of the investigation, they had unearthed sufficient facts to satisfy them that there had been murders, to satisfy them of the method of commission of the murders, and they had narrowed down the suspects to one. I say nothing about the correctness of their suspicion. I say only that on the facts that they had at the time and before the intended interviews were undertaken, the suspicion in my view was not unreasonable.

The position at the end of that third day (March 24) was this: the Police in teams of two were to interview three of the other members of the nursing team in the early morning and thereafter, Staff Sergeant Press and Sergeant Warr would interview Miss Nelles. There is no doubt in my mind that Staff Sergeant Press' suspicions were such that he intended, upon the conclusion of the last interview, in the absence of some information received to allay those suspicions, to arrest Susan Nelles for the murder of Justin Cook.

(c) The Nurses

The Head Nurse for Ward 4A was Elizabeth Radojewski. Under her were four teams of nurses. The team on which Miss Nelles normally served (often referred to as the

Trayner team) consisted besides Miss Nelles of the following:

Phyllis Trayner, R.N. (Team Leader)
Sui Scott, R.N.
Marianna Christie, R.N.A.
Janet Brownless, R.N.A.

Actually Miss Brownless was not a regular member of the team during the epidemic period. She did not join the Hospital staff until August 25, 1980, and for some time thereafter was undergoing introductory or orientation training. From the time she did come on duty she worked more often with other teams than with the Trayner team. She was, however, on the team on the long night shifts of March 20-21-22, and was relieved of duty along with the other members of the team on March 22.

As we have seen, Miss Nelles was assigned to Ward 4B on the night Kevin Pacsai died. Regularly, one team served on Ward 4B opposite the Trayner team. That team was known as the Bell team and consisted normally of the following:

Bertha Bell, R.N. (Team Leader)
Mary Anne Bracewell, R.N.
Mary Jean Halpenny, R.N.
Yvonne Lyons, R.N.A.

From time to time Susan Reaper, R.N. was a member of the team replacing one of the other nurses. The Head Nurse for Ward 4B was Mary Costello.

On Wednesday, March 18, Susan Nelles was off duty and at her parents' home in Belleville. There, she received a telephone call from Mrs. Radojewski informing her that there was to be an inquest into the death of Kevin Pacsai, who had been found to have a high digoxin level. Mrs. Radojewski advised her to make notes of what she remembered of the baby's last night. Miss Nelles was not too happy about the interruption of her holidays and did not actually make any notes until after she returned to Toronto the following day.

She returned of course to the traumatic events of the weekend March 20-22 and the deaths of Allana Miller and Justin Cook. On the Sunday she was advised by Mrs. Radojewski not to come in that night and on Monday she was told by Ms Bell of the steps taken on the wards, i.e., supervisors and no further elective admissions, all in accordance with Dr. Carver's memorandum of that day (see ante page 154). Mrs. Radojewski at some time, probably on Monday, said to her something about going to the Registered Nurses' Association of Ontario for assistance. To that, Miss Nelles replied that it was unnecessary as she had a room-mate who would help.

The room-mate was a third-year law student who seemed also to have had both a brother and fiancé in the law. The room-mate advised her that if questioned she should have legal counsel, and early Wednesday morning, March 25, the brother telephoned with the names of two lawyers who were familiar with nursing matters. Miss Nelles wrote these names on a slip of paper and put them in her robe.

In the meantime, in consequence of the many unexplained changes going on on the wards, a meeting of nurses was held at Mrs. Radojewski's home on Monday. Miss Nelles and all of her team except Mrs. Christie, as well as the Bell team, were in attendance. Although Mrs. Radojewski and Ms Costello knew that the investigation had broadened, because they had been interviewed by the Police that afternoon, they had been pledged to secrecy, and the conversation for the most part centred on Kevin Pacsai. Miss Nelles expressed no concern as she had remembered checking the dosage of digoxin given to that baby with Mary Jean Halpenny, a fact that was confirmed at the meeting by the latter.

This meeting came under considerable suspicion from the Police when they heard about it many days later. They associated it with what they considered withholding of information by some of the nurses, and the tendency to protect their own. I have no doubt that there was a disinclination by some nurses to say anything that might condemn a colleague - it was apparent even at the Commission - but I put it down not to conspiracy but to a common concern about the events taking place at the Hospital over which they seemed to have no control and little understanding. They felt they were being harassed unfairly and unnecessarily and they thought it appropriate to form a common cause. In their defence further, at that meeting only Mrs. Radojewski and Ms Costello knew of the Police investigation; it never occurred to any of the others that any babies were being poisoned. Indeed, to many of them, it is a foreign thought even today.

14. THE ARREST

As we have seen, the Police plan was to interview the four members of the Trayner team who had been on duty the previous weekend, with Susan Nelles being interviewed last. What happened then would depend in part on the earlier interviews, but unless something happened there, or some explanation was given by Miss Nelles herself, the arrest was inevitable.

In the early morning of Wednesday, March 25, the Police teams were dispatched: Staff Sergeant Sangster and P.C. Murray went to interview Phyllis Trayner, Sergeant Barbour and P.C. Hulcoop went to interview Janet Brownless, and Staff Sergeant Press and Sergeant Warr went to interview Marianna Christie. The intention was that the last pair would then check with the other investigators, and armed with all the information so gleaned, would attend upon the prime suspect.

And that is what they did; they concluded their interview with Mrs. Christie at about 10:30 a.m., met with Staff Sergeant Sangster in the lobby of Mrs. Trayner's apartment house, and telephoned Sergeant Barbour at Janet Brownless' home before proceeding to that of Susan Nelles.

I think we should now reflect upon what additional information the visitors to Miss Nelles had obtained that morning. From Mrs. Christie they learned that Mrs. Trayner had relieved Susan Nelles at some time on the night Justin Cook died. Mrs. Trayner had also told her interviewers that she had relieved Susan Nelles for coffee breaks and dinner, and it was apparent from her statement that the nurses from Ward 4B regularly came to see Ward 4A patients. Staff Sergeant Sangster relayed this information to Staff Sergeant Press. As a result, the officers knew several people had been in Justin Cook's room that night. Mrs. Trayner's last relief break was over by 3:00 a.m. according to her statement. Both Mrs. Christie and Miss Brownless stated they had been in Justin Cook's room within fifteen minutes of the onset of his critical symptoms. They both left when Susan Nelles was there with Justin Cook after Phyllis Trayner's last relief. They, of course, had been attending not to Justin Cook but to babies under their care.

None of this information appears to have affected the resolve of Staff Sergeant Press or Sergeant Warr to arrest Miss Nelles. I think there are two basic reasons for that resolve: (a) their great concern was with the death of Justin Cook. So long as the fifteen minute interval between administration and onset of critical symptoms, and the presence of Susan Nelles alone with Justin Cook during that period remained unchallenged, Susan Nelles had exclusive opportunity; and (b) their colleagues, whose judgment they trusted, had found the other nurses open and co-operative and had expressed a belief in their innocence.

In short, the Police remained determined to arrest Susan Nelles unless something developed at the interview with her to change their minds or at least to give them serious concern.

There was no such development. At the interview at Miss Nelles' home, Staff Sergeant Press did the interviewing and Sergeant Warr the recording. The exchange was as follows:

> Q. [Press] Are you Susan Nelles?
> R. [Nelles] Yes.
> Q. We're Police Officers. I'm S/Sgt. Press and this is Sgt. Warr. May we come in and speak with you?
> R. Certainly.
> Q. [Nelles] Are you from the Coroner's Office?
> R. [Press] Yes. We were called by the Coroner to investigate certain deaths, one of them being baby Justin Cook. In connection with his death, I would like to ask you a few questions but first I must tell you that .... [The caution follows. This is the caution referred to ante at page 180].
> Q. [Press] Do you understand that?
> R. [Nelles] Yes. My roommate is a law student.
> Q. Do you understand that you may be charged?
> R. Yes. I believe I know what my rights are.
> Q. OK. Justin Cook died of an overdose of digoxin, a drug he wasn't supposed to have. We believe you gave him the drug, and we'd like to know why. Do you wish to give any explanation for his being given digoxin?
> R. I think I want to speak to a lawyer.
> Q. Do you have any particular lawyer that you want to call?

R. Yes. I have two names here.
[She produced the slip of paper from her robe.]
[Press] Okay. You can make a call from the Police Station.

Q. [Nelles] Why the Police Station?

R. [Press] Because I am arresting you on a charge of murder and I would again caution you. [The usual complete caution was here given.]

Q. [Press] Do you understand that?

R. [Nelles] Yes. I'll wait to speak to a lawyer.
[Press] OK. Please get dressed and we'll get on our way.

The actual arrest took place at about 11:45 a.m.

I make certain comments about the arrest: On the surface, it appears that Susan Nelles was placed in an impossible position by the opening gambit of the Police. She was accused, in effect, of murder and was asked to explain herself. Under our law and our tradition, an accused person need never answer the accusation, and yet if she did not answer, she was facing arrest. She testified that if she had known that she would have answered. But she did not know that until the arrest had taken place.

But it is not as simple as that. The Police too were faced with a dilemma. They believed Susan Nelles had killed Justin Cook, and they were, as they put it, 90% sure they would arrest her. That being their state of mind, her answers in the interview would be inadmissible in evidence under our law unless a caution had been administered. No police officer wants to interview a suspect under these circumstances. Accordingly, a warning was essential, and the warning inhibited the answer. Under our law and tradition also, an accused person has a right both to refuse to answer questions from the police (or anyone else), and to request legal advice. That is, in effect, what Susan Nelles did. Neither the law nor anyone acquainted with our tradition will condemn her for that. But we must be realistic. A police officer already suspicious to the point of conviction is not going to become less so when the suspect declines to answer and produces from her pocket the names of two lawyers she wishes to consult. We have seen why she had those names so readily available but no explanation was given (or indeed asked for) at the time.

One further matter. I think it is clear that Staff Sergeant Press did not arrest Susan Nelles because she said she wanted to see a lawyer. He arrested her because of his previous high suspicion and because she gave him no explanation to allay that suspicion.

15. THE EVENTS IMMEDIATELY SUBSEQUENT TO THE ARREST

The Police took Miss Nelles first to #14 Division Police Station where she was given an opportunity to telephone a lawyer. She called Ms Elizabeth Symes who was one of the lawyers recommended to her by her room-mate's brother. Ms Symes had little or no criminal experience and was able (after some difficulty) to reach a lawyer who had, Mr. David Cole. They met at the Police Station, and after some preliminaries, were permitted to see Miss Nelles alone. There, Ms Symes took notes while Mr. Cole spoke to the accused. He did not ask her for her story, indeed he did not wish to hear her story; instead, he told her that she should assume that the interview was being overheard by the Police. He also told her that she had a right to remain silent and should do so whenever she was unsure of any factual matter. But on the other hand, if she failed to answer any questions at all she might antagonize the Police, who in that event might react by "getting rough" with her. Also according to Ms Symes' notes, confirmed by her (which I accept), he said it was inadvisable for either him or Ms Symes to be present during the subsequent Police interview. (Actually, it appears that he had asked Staff Sergeant Press if Ms Symes could be present and that request was denied). Mr. Cole and Ms Symes thereupon left their client to the interview.

I was disturbed by the advice given. It is not outside my experience that police eavesdrop upon private conversations without authority, but it is, certainly in the case of conversations between solicitor and client, extremely rare and extremely reprehensible; indeed, Mr. Cole conceded that he had no experience of it happening with the Metropolitan Toronto Police Homicide Squad. I am satisfied, however, that Mr. Cole was genuinely concerned about the prospect, and he is not alone in the defence bar in that concern.

I was also disturbed about the ambiguity of the advice given to Miss Nelles of how to conduct herself in the subsequent interview. Defence counsel must advise according to the circumstances of the particular case but in my view it would have been better to tell her to say nothing. As he did not have her story he could not advise her to tell all. Again, I must not blame Mr. Cole. His understanding of the law (shared I am told by some counsel, both for the defence and the Crown) is that there may be some impropriety in some circumstances in advising a client not to answer a police officer's questions. It is not my understanding of the law (certainly if the person asked is accused of a crime), but I am not here to resolve that problem.

The result of the advice, however, is shown in Sergeant Warr's verbatim record of the subsequent interview.

Q. [Press] Do you feel all right?
R. [Nelles] Uh huh.
[Press] If I talk slowly it is because he is recording what we say.
R. Uh huh . . .
Q. I wish to speak to you about baby Justin Cook. Are you familiar with the name Justin Cook?
R. Uh huh.
Q. Would you say "yes" or "no"? He can't record "uh huh" too well.
R. Yes.
Q. Were you on duty on March 20, [19]81, between [the] hours of 1900 and 0300 hours?
R. What's today?
[Press] This would have been Friday night.
R. Yes.
Q. Is that your writing? (Shows piece of paper from file).
R. Yes.
Q. And the baby was received in the Ward directly from the parents?
R. I believe so.
Q. Were you on duty March 21, [19]81, commencing [at] 1900?
R. Yes.
Q. Were you directed to provide constant care to baby Cook?
R. Yes.
Q. Which room was baby Cook in?
R. I don't wish to answer that at this time.
Q. Did you attend to baby Cook?
R. I don't wish to answer that at this time.
Q. Is this your writing? (Showing piece of paper from file).
R. Yes.
Q. With your duties in looking after baby Justin

Cook, were you required to give him any medication?

R. I don't wish to answer that at this time.

Q. Did you know he wasn't supposed to receive digoxin?

R. I don't wish to answer that at this time.

Q. Do you wish to tell me if anything unusual happened to baby Justin Cook during the early morning hours of Sunday March 22, [19]81?

R. No. I don't wish to answer that at this time.

Q. Do you wish to tell me who was on duty with you on Ward 4A during this same period of time?

R. No - I don't wish to answer that at this time.

Q. Do you wish to tell me if anyone other than yourself tended to baby Justin Cook or gave him any medication?

R. No.

Q. No what?

R. No, I don't wish to answer that at this time. [Press] I'd like to speak to you now about baby Allana Miller.

Q. Were you on duty during the evening of Friday, March 20, [19]81?

R. Yes I was.

Q. Commencing at 1900 hours?

R. Yes.

Q. Do you wish to tell me what your assignments were during that shift?

R. No I don't.

Q. Who was on duty with you?

R. I don't wish to answer that at this time.

Q. Do you wish to tell me if at any time during your shift you came into contact with baby Allana Miller?

R. Yes.

Q. Do you wish to tell me at what time you came into contact with baby Allana Miller?

R. No, I don't wish to answer that at this time.

Q. Do you wish to tell me if you administered any medication to baby Allana Miller?

R. No - I don't wish to answer that at this time.

Q. Do you wish to tell me if anyone else administered any medication to baby Allana Miller?

R. No - I don't wish to answer that at this time.

Q. Is this your handwriting? (Shows piece of paper from file).

R. Yes it is.
[Press] Now I'd like to deal with the subject of baby Kevin Pacsai.

Q. Do you wish to tell me if you were on duty during the evening of Wednesday, March 11, [19]81, commencing at 1900 hours?

R. Yes I was.

Q. Do you wish to tell me what duties you were assigned to for that shift?

R. No I don't wish to answer that at this time.

Q. Who was working with you?

R. I don't wish to answer that at this time.

Q. Do you wish to tell me if you came into contact with baby Kevin Pacsai at any time during that tour of duty?

R. Yes I did.

Q. Do you wish to tell me what time?

R. No.

Q. Do you wish to tell me if you administered any medication to baby Kevin Pacsai?

R. No I don't wish to answer that at this time.

Q. I'm showing you a photostat copy of a document marked "Progress Notes". Does that appear to be your handwriting?

R. Yes it does.

Q. Is there anything you wish to tell me about the deaths of babies Cook, Miller or Pacsai?

R. No.

Q. We are also investigating the death of a baby Janice Estrella on January 11, [19]81. Do you wish to answer any questions with regard to the death of that baby?

R. No I don't wish to answer any more questions.

Miss Nelles was afraid to answer any question if not certain of the answer and yet afraid to refuse to answer all questions for fear of abuse by the Police. She interpreted Mr. Cole's advice as suggesting the possibility of physical abuse, a truly monstrous prospect - Miss Nelles is a slim young woman under five feet tall. In his testimony Mr. Cole said he had not intended by the reference to the Police "getting rough" to indicate any violence, but that is the way she interpreted it.

The Police were nonplussed by the interview. They could not understand why she was choosing to answer some questions and declining to answer others which were relatively harmless - they had been told by Mr. Cole only that "she had been advised of her rights". They reached the conclusion that Miss Nelles was fencing with them which did nothing to shake their conviction of her guilt.

Another consequence of the advice was that Judge Vanek was persuaded at the Preliminary Inquiry to admit the statement because it was not a blanket refusal to say anything. He did, however, state - and in my respectful opinion very properly - that he doubted very much the relevance of the statement to any issue before the Court.

I think I can very briefly deal with the events prior to the laying of three additional charges on March 27. Miss Nelles was taken to Court, where the case was remanded to March 26, and thence to the West Detention Centre. The Police commendably took her there themselves rather than have her face her fellow prisoners. They also arranged for separate and private accommodation for her in the Centre. There she remained in custody until she was released on bail the following Monday, March 30. The bail was unopposed, both by the Police and the Crown.

Except for the most hardened criminals it is always a traumatic event to be imprisoned, and in the case of Miss Nelles who had no previous dealings with the law, I am sure it was particularly so. But more important for her future was not what was happening inside the Detention Centre, but what was happening outside.

First of all, her family had been notified of her arrest, and they and Mr. Cole had retained Austin Cooper, Q.C. to lead the Defence. He in turn assigned as Junior Counsel, Mr. Mark Sandler. Mr. Cole undertook the arranging of bail and remained associated with the Defence throughout the Preliminary Inquiry, but not on a regular basis.

It was contemporaneously decided that Mr. Wiley would be associated with, and led by, his immediate superior, Robert McGee, Q.C., Deputy Crown Attorney for the County of York, in the conduct of the Prosecution. When hereafter I refer to Defence Counsel, I shall mean either Mr. Cooper or Mr. Sandler or both; when I refer to Crown Counsel, I shall similarly mean either Mr. McGee or Mr. Wiley or both. When it is is important to distinguish, I shall refer to Counsel by name.

It will be noted from the interview at the Police Station that the Police were concerned not just with the charge relating to Justin Cook. They were very much concerned with the three other babies as well, namely, Janice Estrella, Kevin Pacsai, and Allana Miller. They were convinced that those three babies had been murdered, and, although the cases were not as strong, they firmly believed she was responsible for those as well and they were expecting to find the evidence that would prove it. Their question was whether to charge her with their murder, or to leave the single charge outstanding, and introduce the other deaths as "similar fact evidence", i.e., use the facts of the other deaths in similar circumstances as evidence in the prosecution against her for the murder of Justin Cook. I think it only fair to the Police to add, that if they were going to charge her with complicity in the other deaths, they realized the charges should be laid before the bail hearing. Otherwise, she would have to be rearrested and go through the bail procedure again.

To resolve the problem, Staff Sergeant Press and Sergeant Warr sought and obtained a meeting with Crown Counsel on the afternoon of Thursday, March 26. After being informed of the state of the investigation, Mr. McGee advised the investigators to lay the additional charges. He was satisfied that there were reasonable and probable grounds, and, if the babies had been murdered, he would have found it difficult to explain to the parents why charges of murder had not been laid. The charges were therefore duly laid the following day. Miss Nelles was brought back into Court to have the charges read to her and remanded back into custody. As I have said, she was released on bail the following Monday, March 30.

16. THE INVESTIGATION TO THE COMMENCEMENT OF THE PRELIMINARY INQUIRY

As I have said, the police investigate; the Crown prosecutes. But investigation and prosecution are not distinct and separate; an investigation does not necessarily end with the arrest; in the course of a prosecution, as in any trial, further investigation is often required. Moreover, the investigating police officers are always available to assist the prosecutor, very often in the court room during the trial.

At the time of Susan Nelles' arrest, clearly the investigation was not complete. Of the 4A/B nurses, only four members of the Trayner team had been interviewed (Sui Scott, who was not on duty the last fatal weekend, was not interviewed until March 30). Obviously, it was necessary, at the very least, to interview everyone who had been present at the critical time for any of the babies whose deaths were the subject of the charges. During the period from the first of April to the end of October, Staff Sergeant Press was generally unavailable, either through sickness, injury, other duties, or holidays, and Sergeant Warr took charge of the investigation. His plan was to have each witness interviewed separately regarding each baby, starting with the last death, that of Justin Cook, and working backwards - on the principle that it was easier to remember recent events, and it would be best to get that recollection while those events remained recent. As a result of the adoption of this policy, the interviews with respect to Janice Estrella did not start until the first of May.

Sergeant Warr himself took thirty-seven statements from doctors, nurses, parents, and others between March 24 and June 17, 1981; Staff Sergeant Sangster took thirteen; Sergeant Barbour took sixteen; P.C. Murray took twenty-one; P.C. Hulcoop took nineteen, and Staff Sergeant Press took seven. There were several other interviews for which no statements are available. Because of the system adopted of dealing with one baby at a time, the total number of persons interviewed in that period was not as great as the number of statements, but it still represents a prodigious effort. I do not intend to duplicate that effort by summarizing those statements, but I think I can briefly deal with the effect as follows.

(a) It became increasingly apparent that the Police understanding of separate wards without any staff interchange was fallacious. Time after time the nurses reported going to an adjacent ward, either to care for a patient or to visit another nurse. There were specific instances with both Allana Miller and Kevin Pacsai. With Justin Cook and Janice Estrella there were instances of visits, but since those babies were on constant nursing care, they were attended to by the nurse assigned or by her relief.

(b) The case against Susan Nelles for the murder of Justin Cook remained as solid as it was. While, as I have said before, there was evidence that Marianna Christie and Janet Brownless had been in the room within a few minutes of the onset of his critical symptoms, they were not at his bedside. Susan Nelles was and remained there after they had left. Lynn Johnstone, a night nursing supervisor, who in the course of her duties regularly visited the ward, in a statement of March 27 to S/Sgt. Sangster, placed the relief of Susan Nelles by Phyllis Trayner as ending shortly after 3:00 a.m. As we shall see, Ms Johnstone's evidence, in that respect, when she testified at the Preliminary Inquiry varied slightly but importantly. She testified there that she returned to the ward between 3:00 and 3:30 a.m. and the relief ended: ". . . probably between 3:30 and a quarter to 4:00".

(c) No evidence was obtained to show that Susan Nelles was alone with either Kevin Pacsai or Allana Miller at the critical time.

(d) Even accepting the interstitial theory, there was no evidence of opportunity for Susan Nelles with regard to Janice Estrella. That baby was under the constant care of a nurse named Gloria Ganassin during the day shift. In her interview with Staff Sergeant Press, she could not remember Susan Nelles being in the room at any time. Ms Ganassin would have to be relieved during a shift, but she thought the relief nurse could just as well have been an R.N.A., Mary Cooney, because it was unnecessary to give the baby any medication. She further stated that she was

> reasonably sure the I.V. line was restarted by a doctor after going interstitial at 6:00 p.m.

In the result then, despite all the labours of the Police, the case against Susan Nelles remained much the same for Justin Cook and was certainly no better and perhaps worse (from the viewpoint of a committal for trial) for the other babies.

Initially, the Police and the Crown relied on the Hospital doctors and the Coroners, chiefly Dr. Tepperman, for expert medical advice. According to Sergeant Warr, the latter, shortly after Susan Nelles' arrest, not only examined the charts of babies dying in 1981, but also examined the charts of some of the babies who died in 1980 and concluded that nine of the latter babies could have perished from digoxin toxicity. Those were babies Bilodeau, Dawson, Hoos, Turner, Monteith, McKeil, Lutes, MacDonald, and Gosselin. Accordingly, a chart similar to the one prepared for the babies dying in 1981 was made and completed by the end of the first week of April, 1981. The Police noted that Susan Nelles was assigned to all of these babies except Darcy MacDonald. Dr. Tepperman testified that he was shown only a few charts and that his review of all 1980 deaths which occurred on the wards between July and December did not take place until after the Preliminary Inquiry commenced in January, 1982. The conflict becomes important only when we deal with the Stephanie Lombardo matter, which came to the fore on January 26, 1982.

In any event, Dr. Hastreiter was retained by Mr. Wiley after consultation with the Police and Dr. Tepperman in April, 1981, to give independent, expert cardiologic advice. By Report of May 29, 1981, he gave his initial views of the cause of death of twenty-one of the children (the ones noted in the two charts prepared by the Police) and answered certain specific questions. He also updated the Report from time to time, eventually to record his views on all thirty-six ward deaths and certain other deaths which occurred elsewhere in the Hospital. I have incorporated his views, as modified in his testimony, in Chapter 10, and I need not repeat them here. I should, however, mention that he advised Sergeant Warr (who by this time was aware that the interstitial theory was hardly valid) that a slow infusion of digoxin was possible by injection into the I.V. bag. This would extend the time of maximum effect of the drug from one to several hours depending on the rate of flow of the I.V. itself. Dr. Hastreiter also thought that injection into the naso-gastric tubing through which the baby was being fed was a possibility (at the time Dr. Hastreiter was not aware, or did not appreciate, that feeding by naso-gastric tube was not continuous). This seemed to keep the case against Susan Nelles for the murder of Janice Estrella alive. Certainly, Dr. Hastreiter's opinion served to confirm the worst suspicions of the Police that digoxin was the cause of death of many of the children, including Janice Estrella (although he changed his mind regarding this death later). Dr. Hastreiter, in his initial Report, postulated that very large doses of digoxin would be necessary to account for the levels recorded in the four babies whose deaths were the subject of the charges. This, as Mr. McGee pointed out, forever removed for him the possibility of accident because the amounts were too large. It also to him indicated that the killing had to be premeditated.

In the meantime, Crown Counsel were preparing themselves for the forthcoming Preliminary Inquiry scheduled to begin before Judge Vanek on October 13, 1981. As the summer wore on, it became increasingly obvious that the Crown could not be prepared to proceed on that date. There were some subsidiary reasons, but the main difficulty was that Mr. Cimbura, who was examining the hearts, and other organs and tissues of the four babies, and other babies, would not be ready with his final results in time. A formal application for an adjournment was made on September 11. Mr. Cooper opposed the application, but after argument Judge Vanek granted it, initially until the week of January 4, 1982, exacting a "guarantee" from Mr. McGee that the Crown would proceed on the new date. That new date later was fixed at January 11. The extension was much valued by the Crown. Not only was Mr. Cimbura able to complete his tests and experiments, but other experts including Dr. Hastreiter were consulted on many aspects of the case. Mr. Wiley had some misgivings about the adequacy of the Police investigation, particularly into the background of Susan Nelles, but those misgivings seemed to have been set to rest by the time the Preliminary Inquiry commenced. Nevertheless, as the new date approached, some very serious problems were still outstanding.

The first of these, was the resolution of what were termed the "dirty tricks". Starting in August and continuing into October there were well over thirty incidents of a threatening nature directed at either Mrs. Trayner or Mrs. Scott or both. The Police spent a great deal of time in an effort to discover the identity of the perpetrator(s) of the acts. While their efforts were not successful, much testimony was elicited both at the Commission and at the Preliminary Inquiry, initiated in cross-examination by the Defence, with respect to the incidents, and with respect to the identity of the perpetrator(s). There was no consensus as to the latter, and I should not speculate. The incidents included telephone threats directed to "baby killer(s)", "X" marks on apartment doors, motor cars, windshields, and lockers, and propranolol found in soup, salad, and yoghurt. Once again Mr. Wiley did not seem to be satisfied with the sufficiency of the Police investigation.

At some time, before making a formal Report, Mr. Cimbura informed Dr. Tepperman of preliminary tests on the fixed tissue of Jordan Hines, and that these had disclosed the presence of digoxin. It will be remembered that that baby was not prescribed digoxin and Mr. Wiley and the baby's father had a discussion about the matter on November 27. Mr. Hines was very concerned that his son had been murdered and readily consented to (indeed he proposed) an exhumation to enable Mr. Cimbura to do further tests on exhumed tissue. As we know, this tissue also revealed the presence of digoxin. Sergeant Warr, in December, considered laying a further charge (presumably against Miss Nelles) in connection with the baby's death. Many witnesses were interviewed, but in the end it was decided that insufficient information had been obtained to justify a further charge.

In early January, 1982, all concerned were heavily involved in preparation for the Preliminary Inquiry. Mr. Wiley, worried about the case relating to Janice Estrella, asked Dr. Tepperman on January 5 for any other theory that would account for the long delay between Susan Nelles going off duty, and the death of the child. That night Dr. Tepperman studied some articles and other literature on the subject of digoxin, and the following morning reported that in his opinion the dosage could have been given orally and not by I.V. as everyone had previously assumed. If so, the full effect might not be realized for many hours after administration, since as noted earlier, the peak effect after oral administration is achieved much more slowly.

While this theory might have assisted the Crown's case with Janice Estrella, it could hardly do so for the other babies. Indeed, if valid, it would remove the linchpin of the case respecting Justin Cook. No longer would there be exclusive opportunity on the part of Susan Nelles, and the potential suspects would be almost unlimited. Whole new areas would have to be and were investigated because the digoxin could have been mixed with formula many hours before the baby was actually fed.

Needless to say, the Crown was somewhat disturbed by this development and contemplated seeking an adjournment, notwithstanding the "guarantee" given to Judge Vanek. However, Dr. Hastreiter was coming to Toronto on Sunday, January 10, to prepare for the Preliminary Inquiry, and it was decided to await his arrival and discuss the oral administration theory with him. Dr. Hastreiter did not reject it outright, but he still thought the I.V. route the more likely, and it was decided to let the Preliminary Inquiry proceed.

17. THE PRELIMINARY INQUIRY

A Preliminary Inquiry is not, of course, a trial. It is a judicial investigation to determine whether or not there is sufficient evidence to justify putting the accused on trial. That is, it is a protection to the accused to save him the expense and inconvenience of trial where there is not "any evidence upon which a reasonable jury properly instructed could return a verdict of guilty". U.S.A. v. Shephard, [1977] 2 S.C.R. 1067, per Ritchie J. at page 1080.

But the Preliminary Inquiry has, or has come to have, an additional function. By the opportunity to hear and cross-examine upon the evidence tendered by the Crown, the accused gets to know before trial the case the Crown has against him, and thus is enabled better to prepare his defence. In Ontario for the past few years, there has been a Crown policy of "full disclosure" and where that policy is followed, the second function of a Preliminary Inquiry may not be so important. Indeed, a proposal has been made to the Attorney General for Ontario by a Special Committee of Jurists and Lawyers, under the Chairmanship of Mr. Justice Arthur Martin, to substitute certain specified written material for the Preliminary Inquiry. The proposal outlines certain safeguards for the protection of the accused. There was a minority report opposed to the abolition of the Preliminary Inquiry, and to date no steps have been taken to put the proposal into effect.

So long as the Preliminary Inquiry is with us, it has the two functions I have mentioned. We must bear that in mind when considering the progress of the Preliminary Inquiry in the instant case.

The duty of the Crown Attorney is, of course, to present the evidence and to seek in an appropriate case a committal for trial. The duty of defence counsel is to probe that evidence with a view either to showing that it is insufficient for committal, or if a committal must come, to bring about at least the opportunity for a better defence at trial.

In an adversary system such as ours, many conflicts may arise between counsel in the course of any litigation including Preliminary Inquiries. It is to the credit of all Counsel here that very few of such conflicts arose.

First of all, the policy of "full disclosure" was adopted from the beginning. On August 25th, 1980, Messrs.

Cooper and Cole had the benefit of considerable disclosure and on September 2 the Defence was provided with four volumes of the Crown Brief which contained copies of all statements received up to that time. From time to time also, the Defence would ask for (and invariably receive) assistance from the Crown (and through the Crown, the Police) in pursuing evidence that the Defence thought would be helpful to their case. Mr. Sandler said he never felt there was less than perfect co-operation. Mr. Cooper said he had no complaint about the adequacy of the disclosure and had no complaint about the fairness of the conduct of the Prosecution. It was equally clear that Messrs. McGee and Wiley had nothing but respect for the conduct of Defence Counsel throughout. I mention this because there developed and remain fundamental differences of viewpoint between the Crown and the Defence. There was, however, no rancour and no ill will at any time.

As I have said, there was full disclosure of all statements obtained up until September 2. Preparatory to the Preliminary Inquiry, however, many of the witnesses were interviewed by Crown Counsel and further statements received. These in the ordinary course were not given to Defence Counsel. I have no criticism of that; it would be an intolerable burden for the Crown to have to supply further statements to the defence unless those statements contained major variations. There is only one instance where that situation might be said to have prevailed. In her initial statement with respect to Justin Cook, Phyllis Trayner had said that she had completed the relief of Susan Nelles for the lunch break before 3:00 a.m. In a later statement taken November 2, she said: "I relieved Susan who was with Justin, at about 2:30 a.m. I wanted her back at 3:30 a.m.". In a still later statement taken on January 7, 1982, she is reported to have said: "Fed Cook baby. 2:30->3:30". It is certainly quite possible to interpret those later statements as an admission that she (Phyllis Trayner) was with the baby until 3:30 a.m., only fifteen minutes before the onset of critical symptoms. I think that when there is a difference such as that, going to the root of the prosecution's case, where full disclosure is a practice, that difference should be revealed to the defence. I am satisfied on the evidence that neither Mr. McGee nor Mr. Wiley appreciated the importance of the evidence, and that the failure to disclose it was attributable entirely to inadvertence.

When Phyllis Trayner testified, she said she relieved Susan Nelles just before 2:00 a.m. and fed the baby for thirty to forty-five minutes making the approximate termination time only as late as 2:45 a.m. Obviously, had Mr. Cooper had the later statements, they would have been the subject of cross-examination. In all probability, however, it would have been impossible to set a precise time for the end of the relief. At the Preliminary Inquiry Mrs. Christie placed it at 1:45 to 2:00 a.m.; Miss Brownless could not remember any relief at all after midnight, and Ms Johnstone, as I have said (see ante page 196), testified that Susan Nelles was in the nursing station between 3:00 and 3:30 and thereafter some time between 3:30 and 3:45 went back to the baby's room to relieve Mrs. Trayner.

The timing of the relief was, of course, important because on it would depend the exclusivity of Susan Nelles' opportunity. But it was not so important for the purposes of a Preliminary Inquiry. The task of the Judge, I repeat, is to determine the sufficiency of the evidence, not to weigh it and determine its merit or credibility. So long as there was any evidence of exclusive opportunity, the case was, for the purposes of the Preliminary Inquiry, sufficient.

There were two other matters of evidence that came up at the Preliminary Inquiry about which some considerable question might be raised as to their sufficiency. Mrs. Trayner reported a conversation that she had had after the death of Kevin Pacsai with Susan Nelles in which the latter had said words to the effect she was relieved that Kevin Pacsai had died because now, perhaps, the doctors would pay some attention when she or any nurse reported a child sick and in danger. She was referring back to the difficulties she had experienced with the cardiac fellow on the night of the baby's death where he had not treated the matter as seriously as she thought it deserved and had gone home. The other matter was a statement she was alleged to have made after the death of Justin Cook in a reference to the number of deaths relative to the number of nights on duty: "Six out of seven isn't bad" or words to that effect. It was alleged that the two statements portrayed a callous disregard for human life. Judge Vanek thought they portrayed nothing of the sort, that he could draw no inference of guilt from them whatsoever and I entirely agree. The statements could much more sensibly be attributed to frustration and stress. They demonstrate to me nothing evil; at worst they were tasteless or carelessly expressed.

The most important thing that happened in the early part of the Preliminary Inquiry was a re-appraisal of the death of Stephanie Lombardo. On January 26, Dr. Tepperman, in reviewing the charts of the babies for the purpose of summarizing the similar fact evidence, added five to the list of those who might be suspected to have been victims of digoxin poisoning. Amongst these was Stephanie Lombardo. Indeed, Dr. Tepperman said it was a "carbon copy" of the others.

As I have said (see ante page 197), there is a conflict as to whether Dr. Tepperman first saw the chart in January or had seen it (and rejected it as not raising suspicion) back in April, 1981; obviously the Police thought the latter was the fact and for that reason did not refer the question of that death to Dr. Hastreiter and did not investigate the circumstances. I think it was probably a misunderstanding and Dr. Tepperman did not see it until January 26, but when he did see and compare that death with those which were the subject of the Preliminary Inquiry, it became immediately apparent that it might be vital to the Defence for the simple reason that Susan Nelles was not on duty at the time of that baby's death or for some days previously. All concerned, i.e., Crown Counsel, Dr. Tepperman, and the Police, agreed the body should be exhumed to determine if there was digoxin in her tissues. As we know, that is exactly what was found. The preliminary results were available on February 4, 1982, and communicated by Mr. Cimbura to the Crown and the Police. There was every intention of communicating the results to the Defence, but it was decided to withhold the information long enough to enable the Police to re-interview Mrs. Trayner. The first inkling of a possible conspiracy between Susan Nelles and Phyllis Trayner was beginning to take shape and the Police did not want Phyllis Trayner warned by Susan Nelles.

In any event, the interview did take place on February 8 and February 9. The Police were impressed by her, finding her both co-operative and truthful. She did not, however, remember the baby, Stephanie Lombardo, at all, or the night of her death.

This was really the turning point of the Preliminary Inquiry. Up to that point, the theory of the Prosecution was that Susan Nelles had exclusive opportunity to kill Justin Cook and an opportunity to kill the others and there could be only one killer. After the exhumation of Stephanie Lombardo, the theory was no longer tenable. If there were only one killer it could not be she. If she killed Justin Cook there had to be either more than one killer or a conspiracy between her and another. The case was falling apart.

The Crown very properly informed Mr. Cooper of this very important evidence and equally properly determined to call the evidence as part of the Crown's case. They would not, however, agree either to a stay of the proceedings (which would need the consent of the Attorney General), or to a withdrawal of the charge, either of which would have been very satisfactory to Mr. Cooper. Mr. McGee would not agree because he continued to think that he could (and should) get a committal on Justin Cook. He was hopeful but less certain on the others. Mr. Wiley, although he shared Mr. McGee's views on Justin Cook, thought that in light of the Lombardo evidence the chances of a committal on Justin Cook were less than even and on the others very slim. He testified that if they had had the Lombardo evidence in December it was very unlikely that they would have launched into the Preliminary Inquiry.

In any event, the Preliminary Inquiry proceeded. There was an adjournment in the proceedings from February 25 to April 7, and thereafter the Preliminary Inquiry continued to the conclusion of evidence on May 4. I think Judge Vanek's comments on the conduct of Counsel should be recorded. (The punctuation or lack of it, is, of course, that of the Court Reporter):

> I would like to express my appreciation to counsel for their efforts not only have all counsel distinguished themselves by a most excellent performance of their duties but I would say particularly with regard to the Crown that I do appreciate that the Crown tried to put before the Court all of the facts that it could. That includes facts that might tell against the Crown as well -- and for the accused as well as the facts that are probative of guilt in the opinion of the Crown.

> With regard to Counsel for the accused, I have thought throughout that he was particularly helpful in every way that he possibly could be to the Court. I have spoke of this case going for forty-five days, I'm quite satisfied it could have gone much longer except for very good co-operation between counsel and particularly I appreciate that many matters went in by consent that might not necessarily have been put in by consent in the hands of other counsel.

On May 21, Judge Vanek gave Judgment. In his eighty-three page reasons, he described the layout of the wards, the duties of nurses and doctors, the background symptoms and course of treatment of the four babies, and the result of the digoxin tests. He found there was sufficient evidence in each case of death by a massive overdose of digoxin deliberately administered but declined to commit Susan Nelles on any charge for several reasons as follows.

1. The evidence against her was entirely circumstantial; there was no direct evidence of her participation in any of the deaths.
2. There was opportunity to administer the overdose to babies Cook, Pacsai, and Miller but not exclusive opportunity. There was no opportunity at all to administer it to Janice Estrella.
3. The expert evidence fell short of establishing a precise time of administration.
4. The proposition that one person was responsible for all the killings was a reasonable one, and the accused could not be responsible for the deaths of Janice Estrella or Stephanie Lombardo.
5. The utterances and conduct of Susan Nelles did not give rise to any inference of guilt, or even of aberrant personality.
6. The statements of the accused to the Police merely reflected her exercise of her

> undoubted rights to remain silent and to consult a lawyer.

For these and other reasons - not in my view so important - he discharged the accused on all four counts.

18. THE JURISDICTIONAL QUESTIONS

I return now to the problems I adverted to in Chapter 2 (see particularly *ante* page 6). What conclusion was I entitled to draw, what comment was I entitled to make, in my Report on Phase II? At the end of the evidence I confess I was no closer to the answers and I posed the following four questions to Counsel:

> (1) Am I entitled to report that the Police or the Crown should or should not have arrested, charged, commenced or discontinued [sic] the prosecution of Susan Nelles?
>
> (2) Am I entitled to report that the Police, the Crown, Susan Nelles or others acted rightly or wrongly in general or in any particular in connection with such arrest and prosecution?
>
> (3) Am I entitled to report that I, knowing what I now do, would or would not, arrest, charge or prosecute Susan Nelles?
>
> (4) Am I entitled to recommend for or against compensation for Susan Nelles?

I must now answer these questions before I proceed to my conclusions. The problem is, of course, always to reconcile the apparently conflicting elements of paragraph 4) of the Terms of Reference. For convenience, I repeat the paragraph as amended by the May 24, 1984, Order-in-Council:

> 4) to inquire into, determine and report on the circumstances surrrounding the investigation, institution, and prosecution of charges arising out of the deaths of the above mentioned four infants; and, *without restricting the generality of the foregoing, the Commissioner may receive evidence and submissions and comment fully on the conduct of any person during the course of the investigation, institution, and prosecution of charges arising out of the deaths of the above-mentioned four infants, provided that*

> such comment does not express any conclusion of law regarding civil or criminal responsibility. (amendment underlined).

I shall deal with questions (1) and (2) first. The conflict is between "comment fully" and "provided that such comment does not express any conclusion of law regarding civil . . . responsibility".

Mr. Lamek has pointed out that Mr. Sopinka's action on behalf of Miss Nelles asserts causes of action which are at present unknown to our law, but may eventually become part of our law. Indeed, it may be still possible for an amendment to be made that will assert further causes of action now unknown but later to be recognized. Am I to be guided by the present law or that which is asserted in the action? He argues that I should take a middle course recognizing that the causes of action most likely to be recognized depend on malice by the Police or by the Crown, and perhaps negligence by the Police. To avoid transgression, I should therefore not comment in any way so as to express a conclusion of law as to the presence or absence of malice. As negligence involves not only carelessness but also a duty of care, if I avoid defining the latter, I may comment as fully as I deem appropriate upon the carefulness or adequacy of the investigation and prosecution.

Mr. Sopinka supported this position and went further and said that even if I were to find no carelessness or inadequacy on the part of the Police or the Crown, it would not be a finding of civil non-liability, because I would be using my own standards, not those of law or any recognized legal standard. No other counsel who addressed the matter dissented from that view.

I still harbour some doubts on the matter, but in light of the unanimously recommended course of action, I shall answer questions (1) and (2) affirmatively and in expressing my views I shall avoid any reference to "malice" or "duty" or "standard of care". Any standards that I rely on will be my own. Malice is a legal concept sometimes defined as involving a wrongful act done intentionally without just cause or excuse. I find it difficult to conceive of anyone acting carefully and adequately and, at the same time, maliciously but I assume it is possible and I shall make no comment on that aspect of the conduct of the Police or the Crown. Much argument was addressed to the legal concept of "reasonable and probable grounds" set forth in Section 450 of the Criminal Code, and I have made some reference to it in the recitation of the evidence (see ante pages 180-181) but I will ignore the body of law on that subject lest it be taken that I am adopting a legal as opposed to a personal standard.

Finally, in adopting this course, I take some comfort from the fact that in determining the propriety of the conduct of the Police or the Crown no constitutional question arises. As I have pointed out, the Court of Appeal did not confine their reasoning to criminal responsibilty only but they did note (infra, Appendix 3, pages 9-11) the constitutional problem that might arise if this Commission were entitled to identify the killer(s). Criminal law and criminal procedure belong in the Federal sphere of jurisdiction.

Mr. Lamek also urges a "yes" answer to question (3) but a "no" answer to question (4). He points out that question (3) is more than just a "hindsight" question, that is, whether now, with all the benefit of everything I have heard and learned, I would consider the arrest or Prosecution appropriate. The first two questions are different in that they are based on what the Police or the Prosecution knew or should have known at the relevant times. He argues that the answer to question (3) cannot affect anyone's civil responsibility because that responsibility must be tested as of the date the action was taken. It does not affect criminal responsibility because the test is whether there was sufficient evidence to justify a charge, not whether the accused was guilty or innocent. He argues for a "no" answer to question (4) upon the ground that any decision for or against compensation would be taken as an indication of my belief in the innocence or guilt of Susan Nelles.

Mr. Sopinka and Mr. Hunt both agree to the affirmative answer to question (3), but they would also answer "yes" to question (4). Mr. Sopinka says that compensation or no compensation is a logical result of the answer to the first three questions. Finding one way or another need not involve a belief in innocence or guilt but only that the charge and prosecution should not have been brought or pursued then or now. Mr. Hunt for the Attorney General specifically asked me on behalf of his client to give my opinion for or against compensation not as an indication of guilt or innocence, responsibility, or lack of it, but based upon: ". . . the unique circumstances of the entire series of events that led to the calling of this Royal Commission . . . and that have been examined over the past fifteen months at this Royal Commission". It may have no legal bearing or consequence but I cannot be uninfluenced by the fact that the two parties most concerned about compensation, i.e., the person to compensate, and the person to be compensated, both have asked me to decide the question. Mr. Hunt asks me to state simply whether or not Susan Nelles should be compensated; he does not apparently want me to go further to determine the amount or nature of compensation. I think that if I go into the matter at all I should give some guidelines in the hope of facilitating the resolution of the dispute.

Mr. Percival and Mr. Strathy argue for a "no" answer to both questions (3) and (4). Mr. Percival's position is very simple. He submits that the Court of Appeal (in a Judgment which rejected his submissions to the contrary) has forbidden me to name the perpetrator(s). Any answer to questions (3) and (4) will be taken as inculpating or exculpating Susan Nelles. If I cannot name the killer(s) directly, I cannot name them indirectly by exonerating others. Mr. Strathy takes the same position. In particular, he argues that a finding for compensation would: ". . . unquestionably be viewed by the public as a determination that she was innocent". I pause here to note that Mr. Strathy's argument has the great advantage of consistency. He is the only one of the four Counsel concerned who has taken the same position in both Phases. He wishes to limit my finding in Phase II just as he wished to limit it in Phase I. Mr. Percival has reversed his position because of the Court of Appeal decision. Messrs. Hunt and Sopinka, who argued for my silence in Phase I, now argue that their victory in Phase I in no way compels me to silence in Phase II. It is a topsy-turvy world.

If we examine again the Terms of Reference we see that paragraph 3) requires me "to inquire into and report on and make any recommendations with respect to . . ." and paragraph 4) requires me "to inquire into, determine and report on the circumstances surrounding . . . ". I do not think the different form given to the two Terms of Reference can resolve the matter. I can find no authority on the subject and was offered none but I have come to the conclusion that I should interpret paragraph 4), even without specific reference thereto, to include permission to make appropriate recommendations arising out of the circumstances found. I can scarcely imagine that the Attorney General, whose office drafted the Terms of Reference, would have asked me to make the recommendation if it had been intended that I should not have the power.

It is obvious that I had, and still have, very serious doubts as to the right answers to these questions. In these circumstances one might consider a further application to the Divisional Court for an answer. Only Mr. Strathy was in favour of that solution and it does not appeal to me. There is first of all a jurisdictional question. The Statute (the Public Inquiries Act, R.S.O. 1980, Chapter 411, Section 6) entitles a Commissioner to state a case to the Divisional Court when "the authority . . . to do any act or thing proposed to be done or done . . . is called in question . . . " (underlining my own). It is certainly arguable that until I have decided which way to proceed (which I have not done until now), the Divisional Court would decline to answer the stated case. I also contemplate that there would be great difficulty in stating the case so that the Divisional Court could make an intelligent decision on whether or not my proposed answer would breach the Court of Appeal's injunction without stating the precise answer I propose, which might, by itself, breach that injunction. But even if these hurdles could be overcome and the stated case proceeded, there is no assurance of a final answer within the reasonably foreseeable future. An appeal would be almost inevitable with the strong and diverse positions taken. A delay of many months, perhaps years, would go a long way to destroying whatever value this Report might have. I must, in the appropriate vernacular, "bite the bullet" now.

I have decided to answer both questions (3) and (4) in the affirmative. However they are answered, they cannot affect anyone's civil responsibility because question (3) is indeed more than just a hindsight question, and question (4) is not concerned with civil liability at all. It is simply a consideration of whether, in my view, Susan Nelles should be compensated. Under our law, there is no responsibility for, nor entitlement to, compensation when the accused is discharged at the Preliminary Inquiry. There may or may not be a claim for false arrest or malicious prosecution, but that is not what I am considering. Nor can the answers to either question mean that Susan Nelles is guilty or innocent; nor can it be reasonably taken that I have an opinion one way or the other. As I have said, the Preliminary Inquiry is designed not to determine guilt or innocence, but to determine whether there is sufficient evidence to justify the accused's committal for trial. A policeman should not arrest, a Crown counsel should not prosecute, unless he believes a committal is likely to result. But a policeman may suspect guilt and yet stay the arrest and a Crown counsel may decline to prosecute with a similar suspicion. Similarly, a policeman may legitimately arrest and the Crown may legitimately prosecute without an absolute conviction of guilt. What they need is a reasonable belief that the evidence is sufficient or will be sufficient for a committal. It is not up to them to determine guilt or innocence. That is for the Judiciary (or in some cases, including murder, the Jury). Their duty is, when the evidence appears to justify it, to bring the accused before the Judiciary for the decision.

We like to think that our judicial system is as good as we can make it. But we all know that sometimes innocent people are convicted and, more often, guilty people go free. All that anyone can legitimately take from my answers to all of the questions is that Susan Nelles should or should not have been arrested or prosecuted then, or should or should not be arrested or prosecuted now, because the evidence available did or did not, does or does not, justify that action. In my view that is what the Terms of Reference contemplate my deciding; in my view also, it is not forbidden by the Judgment of the Court of Appeal.

19. THE CONCLUSIONS ON PHASE II

(a) The Standards

Before I proceed to comment on the conduct of persons involved in the investigation and prosecution, I should set out the rules of conduct that I shall apply. As I have said in the last Chapter, those standards must be my own and not those of law, but I hope that my readers will find my standards not widely different from theirs.

First of all, as the oldest cliché tells us, no one is perfect. Indeed, most of us are a good deal less than that. But people should be reasonably competent to perform the tasks they have undertaken. If they are not, they can reasonably expect to be criticized. They must set themselves reasonable standards of conduct in relation to their fellows and adhere to those standards. But I place their duty no higher than that. No one is infallible and no one is obliged to be brilliant or heroic or altruistic however much we may admire those qualities. With this approach, I consider the conduct of all the persons concerned.

(b) The Hospital and the Doctors

I have no mandate to consider the conduct of anyone before the investigation started, and it did not start at least until the Coroner's office was notified on March 12. It did not start in earnest until the Police were brought in.

Dr. Costigan's action in taking Kevin Pacsai's antemortem blood sample to the Biochemistry Laboratory on March 12, and his reporting of the assay result to Dr. Carver on March 17 or 18, can only be commended. From that time on, it is difficult to find fault with the Hospital or its medical or administrative staff. It is suggested that someone should have told the Coroners and Police of Allana Miller's death at the meeting at the Coroner's office on March 21. That information might have prompted earlier Police action, but it is to be remembered that the digoxin levels for that baby were not known until 8:00 p.m. that day, and the Coroner was then immediately notified. None of the doctors at the meeting was suspicious of that death at the time. And if they had been suspicious of the death, they did not suspect murder, and that perhaps explains the assurance at the same meeting that digoxin was "under control". It certainly was not then under control in the sense that it was unavailable to potential killers. They had, however, assured themselves that the concentration and the prescription and dosage for Kevin Pacsai were correct.

The Police were given full assistance from the first. At one period the Police thought the Hospital was withholding information and being less than fully co-operative, but eventually they became satisfied that this was not so. There were indeed problems in communication. The Hospital and the doctors would answer any requests and any question when asked. The difficulty was that in this very special investigation, the Police often did not know what to ask, and the doctors did not know what to volunteer.

(c) The Nurses

The complaint urged in testimony but not pursued in argument against the nurses was that some of them gave the Police and the Crown something less than full co-operation, that some of them withheld information, and that some of them changed their stories between statements and testimony to assist the accused. I am sure the Police and the Crown believed in the validity of their complaint but I must find it unproved. All of the nurses interviewed did give statements. As I have said, (ante page 184) the nurses felt abused by the investigation and many of them felt sympathy with their colleague and friend who was arrested. No misstatements or obstruction have been shown. I am prepared only to say that there was a reluctance on the part of many nurses to say anything that might reflect upon any nurse. I am not prepared to condemn them for it.

(d) The Coroners

The Coroners also have very little to answer. They called in the Police as soon as any possible suspicion of foul play was aroused, and from the moment they learned of Justin Cook's digoxin levels, the whole investigation was turned over to the Police. It has been suggested that they might have ordered the start of the Police investigation immediately after the meeting on March 21.

But we should remember that at that meeting they were considering the deaths of only two babies whose deaths were two months apart. They were assured that digoxin was "under control". They could hardly be blamed for not investigating other deaths earlier when only three of those deaths appear to have been reported to the Coroner.

I do not believe they had anything to do with the arrest of Susan Nelles. The decision was that of Staff Sergeant Presc with advice from Mr. Wiley. The Coroners perhaps could have given the Police pause if they had disagreed with the proposed arrest but I see no reason why they should have done so in the circumstances.

Staff Sergeant Press was certain Dr. Tepperman had seen the Lombardo chart in the spring of 1981 and rejected that death as one that might be suspicious. I accept Dr. Tepperman's evidence that he did not then examine the chart for that purpose. First, the Police had no record that Dr. Tepperman had said the death was "not applicable" as they had for Dion Shrum, Antonio Velasquez, and Jesse Belanger, indicating to me that Dr. Tepperman was not shown the Lombardo chart at that time. Secondly, when he did see it in January he found it not just similar but strikingly so.

By the time Dr. Tepperman was asked his view of relieving the nursing team on the night of March 21, the team was already on duty and their removal would not only be awkward, but difficult to explain. At the time, Dr. Tepperman knew that digoxin was truly controlled and felt sure nothing would happen that night, and the Police investigation would start the next morning. He was wrong as it turned out, but one can only blame him for the decision now with the great help of hindsight.

(e) Susan Nelles

I think by any standard, and certainly by mine, there was nothing wrong in the conduct of Susan Nelles in her dealings with the Police. As I have said, her actions seem to have contributed to her arrest in that her failure to offer a denial or explanation when first approached, and her selective answers at the Police station, seem to have confirmed the suspicion of the Police. We know why she did what she did, but at the time the Police did not. They never did learn until these hearings began.

I repeat, however, that those actions were exercises of her undoubted rights. Police officers cannot expect the assistance of suspects to prove the case against them. No suspect, guilty or innocent, need answer any question put to him by the police. That is so fundamental to our tradition that it need hardly be said. What Susan Nelles did, may not in the circumstances have been wise, and may not have been in her best immediate interest. I think her arrest and charge with respect to the death of Justin Cook were inevitable, and with respect to the deaths of the other three babies probable, even if she had spoken up, and I remind the reader that she was acting on legal advice which is also a fundamental right.

(f) The Police

The complaints against the Police are many and varied, but essentially they come down to the following three things:

(i) they acted with undue haste in the charge relating to Justin Cook, failing to investigate the bases for their conclusions thoroughly;

(ii) they laid the charges with respect to Allana Miller and Kevin Pacsai without any evidence of Susan Nelles' presence with the child at the critical time; and

(iii) they laid the charge with respect to Janice Estrella, knowing Susan Nelles was not in the presence of the baby at the critical time. They relied upon a theory (the interstitial theory) that could not have withstood any serious scrutiny.

I have related the position (see ante Chapters 13-15) facing the Police at the time of the arrest and charges. We have seen also that many of their assumptions proved wrong before the end of the Preliminary Inquiry. We must, however, judge them not on hindsight, but on what they knew and what it was incumbent on them to find out at the time.

When you sift through it all, there were only two essential facts that guided their actions. They believed that Susan Nelles had killed Justin Cook and they believed that there was only one killer. As I have said, I do not find either proposition - I emphasize upon the information they had at the time - unreasonable. The only problem is whether they should have taken more time and investigated further to make sure there was no error. That was, in effect, the advice of Mr. Takach when he was informed of the impending arrest; that advice does not seem to have been passed on to the Police. I realize that this was certain to become a very high profile case; the arrest of a nurse for the murder of babies would have horrendous effects upon her even if it could not be proved, but I still do not think the Police can be blamed for acting when they did, at least with respect to Justin Cook. It might have been different if they were unsure or if their conclusion was unreasonable. Here, no one differed from their conclusion; they sought advice from the Crown before they acted.

The situation is not so clear with respect to the other babies. They had no evidence of exclusive opportunity with any, and very shaky evidence of any opportunity with one. But the second basic belief here came into play. If there was only one killer, and if these babies had been murdered (again a reasonable inference), the killer must be the same as the killer of Justin Cook. I entirely agree that if the Police had been investigating only the other three, they should not have acted so quickly; the evidence simply was not sufficient for two and barely existed for the third. But the charges for those three are really just incidental; the main charge was Justin Cook and if that charge were proven, it made little difference to Susan Nelles whether the others were proven or not. Indeed, as we have seen, the laying of the charges in connection with Kevin Pacsai and Allana Miller did her no harm and the laying of the Estrella charge proved a considerable help.

In summary, I see nothing wrong in the arrest or in the charge relating to Justin Cook. I think the Police acted precipitately with the others, but I can understand and to a certain extent sympathize with their actions. The later charges did no appreciable additional legal harm to Susan Nelles. She was going to be faced with those three deaths in one way or another at the Preliminary Inquiry. It was a close decision; they took advice from the Crown. They were doing their job as they saw it. I attach no blame to them. I can only commend them for their treatment of Miss Nelles after her arrest.

(g) The Crown

From the time the charges were laid the Police, while they continued the investigation, ceased to have any control over the destiny of Susan Nelles. That control now lay with the Crown, Defence Counsel, and the Judiciary. For the Crown Mr. McGee was clearly in charge; Mr. Wiley was his junior taking an active part in the prosecution, but the ultimate decisions were not his. Mr. Takach was consulted from time to time and gave advice and other senior Crown officials were informed, but there is no instance where Mr. McGee was directed to take any action or where his decisions were interfered with.

The complaints against the Crown were essentially that they continued the prosecution knowing the following:

(i) that the continuing investigation failed to bring out any evidence placing Susan Nelles exclusively with Allana Miller or with Kevin Pacsai at the critical time;

(ii) that the interstitial theory and the I.V. bag and naso-gastric tube theories that replaced it had become untenable as explanations linking Susan Nelles to Janice Estrella's death;

(iii) that Phyllis Trayner in two statements and Lynn Johnstone in evidence, had destroyed the exclusive opportunity theory with Justin Cook; and

(iv) that the Stephanie Lombardo incident had demonstrated that a murder had taken place for which Susan Nelles could not have been responsible.

Mr. Sopinka has stated these propositions to be self-evident. I will assume their truth for the purpose of argument. Upon that assumption, I can only agree that the Crown was obstinately hanging on to an untenable theory. At the same time, I must commend the Crown for leading the evidence that showed that the theory was untenable.

I think where the complaint falls down is that it assumes the Crown must always see things correctly, and if it fails to do so it is somehow acting improperly. It is said the Crown never wins and the Crown never loses. That may be so, but Crown counsel is a lawyer, and it is in the nature of lawyers to be hard to dissuade from the validity of their case. Mr. McGee testified, and I accept his evidence, that he continued to believe to the end of the Preliminary Inquiry that Susan Nelles was the culprit in the death of Justin Cook, that she was the most likely culprit in the deaths of babies Miller and Pacsai, and that he should obtain a committal for the first and perhaps for the others as well. No doubt Crown counsel should stop the prosecution when he believes the accused is innocent; but equally he must continue it if he believes her guilty. When there is doubt in the midst of a prosecution it is not for the Crown to resolve that doubt; that is for the Judiciary. Mr. McGee left it to Judge Vanek; Judge Vanek resolved that doubt. That is the way our system works.

I come to the end then, attaching no great blame to anyone; I can put it no better than did Mr. Cooper in a conversation with Mr. McGee after the discharge:

> You did your job; I did mine.
> The Police did theirs; the Judge
> did his. The system worked.

(h) Compensation

The system worked but it exacted a price and that price was paid by Susan Nelles. Should she be compensated? Our law does not require compensation, but I have been asked to give my personal view and, as I have said, I intend to comply with that request.

Before I do so, I should deal with the problem raised in question (3) of the jurisdictional questions. The answer is that knowing what I now do, I would not recommend the arrest or the charge or the prosecution of Susan Nelles for the deaths of any of the babies. Besides all of the evidence I have outlined, much of which was known to Judge Vanek, and brought about his decision, there is now further evidence not available to him. Dr. Kauffman, whose testimony on all matters pharmacological I find most convincing, gave his estimate of the probable time of administration of the overdose of digoxin to Justin Cook. The ante-mortem blood sample was taken about 4:30 a.m., ten minutes after the cardiac arrest and Dr. Kauffman's opinion was that the dosage would have had to be administered at least one hour before that to account for the distribution to tissue. He said further that the time could be as much as two or three hours before; thus bringing the time of administration to somewhere between 1:30 a.m. and 3:30 a.m., during which time Susan Nelles was relieved for close to an hour. Dr. Kauffman also gave his opinion that it was quite possible that the administration of the overdose to Allana Miller took place either into the I.V. line or the buretrol (a medication chamber in the I.V. line controlling the rate of flow) at times when Susan Nelles was not attending the baby. It follows from this that there is not only no evidence of exclusive opportunity in her for the deaths of Justin Cook and Allana Miller, but there is evidence of equally good opportunity in others.

It follows that there was not then in fact sufficient evidence (although there was legitimate belief that there was) nor is there now sufficient evidence to justify her committal for trial. In a perfect world, she would not have been arrested, charged or prosecuted.

Yet she was, and in the course of it she suffered quite apart from her loss of reputation and her mental anguish, very substantial legal costs. I think she should be compensated for those costs. This was not only a notorious case (and the notoriety continues to this date), but a very unusual one as well. The Preliminary Inquiry occupied forty-one days of evidence and four days of argument. It was extremely complicated and extremely difficult. She needed (and obtained) very good counsel.

I know that her civil claim embraces much more than her legal expenses, but I do not recommend any further payment. As I have said, the law does not now require any compensation in any amount, and any proposals for reform of that law that I have seen do not propose any greater payment than out-of-pocket loss in the absence of long incarceration. I recommend that payment here because the case was notorious, difficult, and lengthy and because there was not then in the result and there is not now sufficient evidence to commit her for trial.

I therefore recommend that Miss Nelles be compensated for her reasonable solicitor and client costs from the time of her arrest to the time of her discharge at the end of the Preliminary Inquiry. She has already been paid her reasonable costs of this Commission. If she lost any income, which I understand she did not, I recommend that she be paid that as well. I am not permitted to make, and I do not make, any comment on the merits of the civil action. I think, however, that it would be unreasonable for her to accept compensation and still pursue her action. She must make her choice.

I think it would be a reasonable condition of this *ex gratia* payment that the civil action of *Susan Nelles v. Her Majesty the Queen et al.* be dismissed on consent without costs.

EPILOGUE

It has taken a long time, but now it is done, and the time has come to thank those who have made it possible. It fell to me to preside at the Hearings and to write this Report, but I am not so immodest - or so foolish - as to think I could have managed it alone.

First and foremost there are the Counsel, Paul S.A. Lamek, Q.C. and Eleanore A. Cronk. I cannot say enough in their praise. They sought out and led all the evidence, one hundred and seventy-six days of it, working nights and weekends and enjoying hardly any holidays. And their work was intelligent, thorough, and very effective. I also thank their clients, who I hope have long memories, for lending them to me.

We had in this Commission the benefit of a computer, something I have never known before either on a commission or at a trial. Dr. Anne Gilmour-Bryson and her assistant Eric Stine put all the evidence on the computer and were able to retrieve it as if by magic for Counsel during the hearings and for me as well after the hearings were over. I never learned to operate the machine but there was no need. Dr. Bryson could always supply the information in very short order. The luxury of not having to pore over my own illegible notes or the mountains of transcripts made my life a great deal more tolerable. There is a great future for the computer in very long commissions (or trials) so long as there is available so efficient a consultant and operator.

Then there was Anita Fineberg a young lawyer who became our very capable research Counsel. On many occasions we benefited not just from her research but from her comment on all the issues.

There were also two young law students who helped us for the first few months - Mark Hayes and James Marsland who had to leave us in September, 1983, to continue their studies. They are no longer young law students; they are now young lawyers.

There were our police advisors from the Ontario Provincial Police, Detective Inspector (now Superintendent) Murray McMaster and Detective Inspector Charles Judson, who did very valuable work in studying, summarizing, and explaining the voluminous police documents. There were Constables Ken McLeod and John McMullen who on the practical side gently but firmly looked after the seating and security of the hearing room.

There were Helen Warburton, Dorothy Kosonic, Brenda Boyd, Phyllis Traversy, and Barbara Lynch (the last on temporary loan from the Royal Commission on the Northern Environment), who so very ably supplied our secretarial needs over the period. There was Bob Kosonic who looked after the office to good effect and with very good will, and there were Mark Davidson and Michael Madarang both high school students, who in the summers helped him out.

There was my old friend Murray Elliott, retired local registrar of the Supreme Court of Ontario, who came back to be Registrar for this Commission. I do not think it would have been possible for anyone to have managed the job and those five hundred exhibits better or with better humour than he.

Finally there was Thomas B. Millar, also a former local registrar of the Supreme Court of Ontario, and even longer retired, who was our Administrator. He very ably saved Counsel and me any concern about the administration of the Commission. We were very grateful, I because I was incapable, they because they had no time.

I hope that I will not lose touch with all these people who helped so much. I suspect they will forgive me when I say also - and fervently - that I hope our next encounter will be a briefer one.

Ontario
Executive Council

Order in Council

APPENDIX 1

On the recommendation of the undersigned, the Lieutenant Governor, by and with the advice and concurrence of the Executive Council, orders that

WHEREAS concern has been expressed in relation to a number of deaths of infants in Cardiac Wards 4A and 4B at the Hospital for Sick Children, Toronto, between July 1st, 1980 and March 31st, 1981, and

WHEREAS concern has been expressed concerning the functioning of the justice system in respect of the instituting and of prosecuting of charges in relation to the said deaths, and

WHEREAS the Government of Ontario is of the view that there is a need for the parents of the deceased children and the public as a whole to be informed of all available evidence as to the deaths and the proceedings arising therefrom, and

WHEREAS it is thought fit to refer these concerns to an inquiry pursuant to the provisions of the Public Inquiries Act, R.S.O. 1980, Chapter 411,

NOW THEREFORE, pursuant to the provisions of the said Public Inquiries Act, R.S.O. 1980, Chapter 411, a commission be issued to appoint the Honourable Mr. Justice S.G.M. Grange who is, without expressing any conclusion of law regarding civil or criminal responsibility:

1) to consider the matters disclosed in the Report of the Hospital for Sick Children Review Committee, chaired by the Honourable Mr. Justice Charles Dubin; the report on "Mortality on the Cardiology Service in a Children's Hospital in Toronto, Canada", by the Center for Disease Control and the Ontario Ministry of Health; and the

evidence disclosed at the preliminary hearing in relation to the charges of murder relating to the death of four infants at the Hospital for Sick Children and, having regard to the undesirability of duplicating unnecessarily the work done by them or unnecessarily subjecting witnesses to further questioning, to draw from such reports and preliminary hearing whatever evidence which he deems relevant and appropriate and to thereby dispense with the hearing of any testimony and production of documents or things that he considers appropriate;

2) to require the summoning of such witnesses as the Commissioner deems necessary to give evidence under oath and to produce such documents and things as the Commissioner may deem requisite to the full examination of the matters he is appointed to examine and to ensure full public knowledge of the completeness of the matters referred to in these terms of reference;

3) to inquire into and report on and make any recommendations with respect to how and by what means children who died in Cardiac Wards 4A and 4B at the Hospital for Sick Children between July 1st, 1980 and March 31st, 1981, came to their deaths;

4) to inquire into, determine and report on the circumstances surrounding the investigation, institution, and prosecution of charges arising out of the deaths of the above mentioned four infants;

AND THAT all Government Ministries, Boards, Agencies and Commissions shall assist the Honourable Mr. Justice to the fullest extent in order that he may carry out his duties and functions, and that he shall have authority to engage such counsel, investigators and other staff as he deems it proper at rates of remuneration and reimbursement to be approved by the Management Board of Cabinet in order that a complete and comprehensive report may be prepared and submitted to the Government,

AND THAT the Ministry of the Attorney General will be responsible for providing administrative support to the Inquiry,

AND THAT Part III of the said Public Inquiries Act be declared to apply to the aforementioned Inquiry.

Recommended ____________ Attorney General

Concurred ____________ Chairman

Approved and Ordered April 21, 1983
Date

____________ Lieutenant Governor

Order in Council APPENDIX 2

Ontario
Executive Council

On the recommendation of the undersigned, the Lieutenant Governor, by and with the advice and concurrence of the Executive Council, orders that

WHEREAS by Order-in-Council numbered OC-1076/83 and dated the 21st day of April, 1983, the Honourable Mr. Justice S. G. M. Grange was appointed a Commissioner under the Public Inquiries Act to inquire into a number of deaths at the Hospital for Sick Children and the proceedings arising therefrom; and

WHEREAS the Commissioner has requested confirmation of the intent and purpose of paragraph four of the terms of reference set out in the said Order-in-Council; and

WHEREAS it is appropriate that the intent and purpose of paragraph four of the said Order-in-Council be confirmed:

NOW THEREFORE, Paragraph four of the said terms of reference be amended to add, after the word "infants" in the said paragraph, the following words:

> "and, without restricting the generality of the foregoing, the Commissioner may receive evidence and submissions and comment fully on the conduct of any person during the course of the investigation, institution, and prosecution of charges arising out of the deaths of the above-mentioned four infants, provided that such comment does not express any conclusion of law regarding civil or criminal responsibility."

Recommended Attorney General Concurred Chairman

Approved and Ordered May 24, 1984
Date
O.C. 1412/84

Lieutenant Governor

APPENDIX 3

IN THE SUPREME COURT OF ONTARIO

COURT OF APPEAL

Howland C.J.O., Brooke, Arnup, Martin and Houlden JJ.A.

IN THE MATTER OF The Public Inquiries Act, R.S.O. 1980, c. 411, and in particular Section 6, Subsection 1 thereof;

AND IN THE MATTER OF The Royal Commission of Inquiry into Certain Deaths at the Hospital for Sick Children and Related Matters

BETWEEN:

SUSAN NELLES, PHYLLIS TRAYNER, THE REGISTERED NURSES ASSOCIATION OF ONTARIO and THIRTY-NINE REGISTERED NURSES AT THE HOSPITAL FOR SICK CHILDREN

Appellants
(Applicants)

-and-

THE HONOURABLE MR. JUSTICE S.G.M. GRANGE, COMMISSIONER

Respondent
(Respondent)

-and-

VARIOUS OTHER INTERESTED PARTIES

John Sopinka, Q.C. and David Brown for the Appellant, Susan Nelles

George R. Strathy and Elizabeth J. Forster for the Appellant, Phyllis Trayner

Frances Kiteley for the Appellants, The Registered Nurses Association of Ontario and thirty-nine nurses

Paul S.A. Lamek, Q.C. and E.A. Cronk for the Respondent, The Honourable Mr. Justice S.G.M. Grange, Commissioner

Douglas C. Hunt and Lucy Cecchetto for the Intervenant, Attorney General for Ontario

Ian G. Scott, Q.C. and Mary M. Thompson for the Intervenant, The Hospital for Sick Children

Barry A. Percival, Q.C. and David S. Young for the Intervenant, The Metropolitan Toronto Police Force

For the parents:

Morris Manning, Q.C. and Stephen M. Labow;

Warren W. Tobias, Jack S. Shinehoft and Fred Shanahan

Appeal Heard:
March 26 & 27, 1984

BY THE COURT:

This is an appeal from the order of the Divisional Court dated the 30th of January, 1984 by which the Court answered in the affirmative a question in a case stated by the Commissioner for its opinion. Mr. Justice Reid dissented and would have answered the question in the stated case in the negative. The case was stated pursuant to The Public Inquiries Act and the question was as follows:

> Was I right in determining that I am entitled in my Report (subject to certain qualifications I have set forth) to express my opinion upon whether the death of any child was a result of the action, accidental or otherwise, of any named person or persons?

The appellants and the Attorney General for Ontario, and The Hospital for Sick Children as intervenors contend that the Commissioner was wrong and that the Divisional Court erred in answering the question in the affirmative. The Hospital for Sick Children supports the judgment of Mr. Justice Reid. The appeal is opposed by the Commissioner himself through his counsel. It is also opposed by the Metropolitan Toronto Police Force and some of the parents who lost their children.

The Hospital for Sick Children at Toronto is one of the great medical institutions in this country. Between July the 1st, 1980 and March the 31st, 1981, thirty-four infant children died in the cardiac wards of that hospital. In addition, one infant died a day after the period which is referred to and another died in the intensive care unit of the hospital shortly after it had been moved from the cardiac ward. All of the children were seriously ill with an identifiable heart condition or disease. An investigation revealed that it was likely some of the children did not die from natural causes in the sense that they did not die from the disease or illness for which they were hospitalized; rather it was suspected that some of them had died because of the administration of an excessive amount of the drug digoxin while they were on the cardiac ward.

When the matter first came to light an investigation by the police resulted in a charge of first degree murder of four of the children being laid against Miss Susan Nelles who was a member of the team of nurses headed by one, Phyllis Trayner, which team was at material times assigned to the duty of caring for the children on the wards where they died. It was alleged against Miss Nelles that those children had died because lethal doses of the drug digoxin were administered by her when those children were patients on the cardiac ward. At the end of the preliminary enquiry the Provincial Court judge refused to commit Miss Nelles for trial and she was discharged. The Provincial Court judge found inter alia that some of the children died as a result of deliberate overdoses of digoxin.

Because of concern as to the practices and procedures in The Sick Children's Hospital at Toronto, a review thereof was authorized by the Lieutenant Governor in Council and undertaken by The Honourable Mr. Justice Dubin and a committee of medical and hospital experts. The review was completed and a report of its findings and recommendations filed, which recommendations to a great extent have been acted upon. But that report did not deal with the cause of the death of the thirty-six children which is the matter this inquiry was concerned with. Indeed that subject was specifically exempted from the terms of reference of Mr. Justice Dubin and his colleagues.

An investigation was carried out jointly by the Ontario Ministry of Health and the United States Center for Disease Control. It dealt with the cause of the deaths in question here. This was the second of two reports which had been prepared on this question.

After considering the last-mentioned reports, the findings of the Dubin committee, the evidence from the preliminary enquiry conducted into the charges against Miss Nelles, the Attorney General for Ontario concluded that there would be no further criminal charges laid at that time against any person or persons for there was insufficient evidence to support them. However, responding to the requirement of accountability to the public, the Attorney General determined that there should be a Royal Commission of enquiry to look into and report publicly on the circumstances surrounding the deaths of the children and the subsequent criminal proceedings against Miss Nelles. Accordingly, on the 21st of April, 1983, the Lieutenant Governor in Council ordered that a commission be issued to the Commissioner. The Order-in-Council is set forth in full.

> WHEREAS concern has been expressed in relation to a number of deaths of infants in Cardiac Wards 4A and 4B at the Hospital for Sick Children, Toronto, between July 1st, 1980 and March 31st, 1981, and
>
> WHEREAS concern has been expressed concerning the functioning of the justice system in respect of the instituting and of prosecuting of charges in relation to the said deaths, and
>
> WHEREAS the Government of Ontario is of the view that there is a need for the parents of the deceased children and the public as a whole to be informed of all available evidence as to the deaths and the proceedings arising therefrom, and
>
> WHEREAS it is thought fit to refer these concerns to an inquiry pursuant to the provisions of the Public Inquiries Act, R.S.O. 1980, Chapter 411,
>
> NOW THEREFORE, pursuant to the provisions of the said Public Inquiries Act, R.S.O. 1980, Chapter 411, a commission be issued to appoint the Honourable Mr. Justice S.G.M. Grange who is, without expressing any conclusion of law regarding civil or criminal responsibility:
>
> 1) to consider the matters disclosed in the Report of the Hospital for Sick Children Review Committee, chaired by the Honourable Mr. Justice Charles Dubin; the report on "Mortality on the Cardiology Service in a Children's Hospital in Toronto, Canada" by the Center for Disease Control and the Ontario Ministry of Health; and the evidence disclosed at the preliminary hearing in relation to the charges of murder relating to the death of four infants at the Hospital for Sick Children and, having regard to the undesirability of duplicating

unnecessarily the work done by them or unnecessarily subjecting witnesses to further questioning, to draw from such reports and preliminary hearing whatever evidence which he deems relevant and appropriate and to thereby dispense with the hearing of any testimony and production of documents or things that he considers appropriate;

2) to require the summoning of such witnesses as the Commissioner deems necessary to give evidence under oath and to produce such documents and things as the Commissioner may deem requisite to the full examination of the matters he is appointed to examine and to ensure full public knowledge of the completeness of the matters referred to in these terms of reference;

3) to inquire into and report on and make any recommendations with respect to how and by what means children who died in Cardiac Wards 4A and 4B at the Hospital for Sick Children between July 1st, 1980 and March 31st, 1981, came to their deaths;

4) to inquire into, determine and report on the circumstances surrounding the investigation, institution, and prosecution of charges arising out of the deaths of the above mentioned four infants;

AND THAT all Government Ministries, Boards, Agencies and Commissions shall assist the Honourable Mr. Justice (sic) to the fullest extent in order that he may carry out his duties and functions, and that he shall have authority to engage such counsel, investigators and other staff as he deems it proper at rates of remuneration and reimbursement to be approved by the Management Board of Cabinet in order that a complete and comprehensive report may be prepared and submitted to the Government,

AND THAT the Ministry of the Attorney General will be responsible for providing administrative support to the Inquiry,

AND THAT Part III of the said Public Inquiries Act be declared to apply to the aforementioned Inquiry. (Emphasis added)

All of the parties in this Court referred to a statement made by the Attorney General for Ontario in the Legislature at the time the making of the Order-in-Council was announced. In that statement he explained that the order was designed to provide an opportunity for the fullest public knowledge of the circumstances of the deaths and the criminal proceedings which followed them. He emphasised the reason for the limitation imposed upon the Commissioner saying the qualification he "is not to express any conclusion of law regarding civil or criminal responsibility, will serve to protect the interests of any past, present or future litigant in civil or criminal matters". He gave the assurance that if evidence ever became available to warrant the laying of additional charges, such charges would be laid and vigorously prosecuted. And then, dealing with the investigation to be carried out, he said:

> I hope you will recall my statement on February 21 in which I said: "...we as a Government are committed to providing the fullest possible accounting of the events and circumstances that led to this tragedy". That commitment will be met.
>
> The terms of reference for the Commission, a copy of which is attached to my statement, expressly provide that the inquiry is not to involve any conclusion of law regarding to civil or criminal responsibility.
>
> This provision was made to ensure that the Commission would not function or be regarded as a criminal or civil trial. Trial by Commission of Inquiry is a concept totally foreign to the Laws of the province.

While it is understandable that many members of the public would wish to have an inquiry to determine responsibility for the tragic deaths, this cannot be the function of a Royal Commission.

The purpose of a public inquiry is not to attach criminal culpability. It is not a forum to put individuals on trial. The just and proper place to make and defend allegations of crime or civil liability is in a court of law.

In this context, I am reminded of the remarks of an eminent Ontario jurist, Mr. Justice Riddell of the Ontario Court of Appeal, whose observations almost 50 years ago are equally applicable today.

> A Royal Commission is not for the purpose of trying a case or a charge against anyone, any person or any institution - but for the purpose of informing the people concerning the facts of the matter to be enquired into.... The object of a Royal Commission is to determine facts, not to try individuals or institutions, and this consideration is sufficient to guide the Commissioner in the performance of his duty.

This principle is particularly important and relevant here because there have been criminal proceedings in relation to the deaths and civil proceedings are pending with respect to the initiation and prosecution of those charges.

We are very sensitive to the need not to interfere with or undermine the rights of any litigant whose matter is now before the courts or who may come before the courts in the future.

I am advised that there is no precedent for an inquiry of this nature. To our knowledge there has never in Ontario been a Commission of Inquiry into deaths which are thought to have been the result of deliberate criminal acts by a person or persons unknown.

The statement of the Attorney General as to the limitation on the scope of a public enquiry was correct and it is important. A public inquiry is not the means by which investigations are carried out with respect to the commission of particular crimes or, using his words, "deaths which are thought to have been the result of deliberate criminal acts by a person or persons unknown." Such an inquiry is a coercive procedure and is quite incompatible with our notion of justice in the investigation of a particular crime and the determination of actual or probable criminal or civil responsibility.

In *R. v. Hoffman-LaRoche Ltd. (Nos. 1 & 2)* (1981), 33 O.R. (2d) 694 at 724-5, 125 D.L.R. (3d) 607 at 637, 62 C.C.C. (2d) 118 at 147-9, Martin J.A., speaking for this Court, said:

> It is well established that a Province may create provincial agencies such as coroners, fire marshalls, securities commissions and commissions of inquiry and endow them with the power to summon witnesses and compel them to give evidence under oath in an inquiry conducted for a valid provincial purpose, notwithstanding that any witness required to give evidence may potentially be a defendant in a subsequent criminal proceeding: see *Faber v. The Queen and two others*, [1976] 2 S.C.R. 9, 27 C.C.C. (2d) 171, 65 D.L.R. (3d) 423; *Di Iorio v. Warden of Common Jail of Montreal, supra*; *A.-G. Que. and Keable v. A.-G. Can. et al.*, [1979] 1 S.C.R. 218, 43 C.C.C. (2d) 49, 90 D.L.R. (3d) 161; *Report of the Canadian Committee on Corrections*, pp. 67-9.
>
> The investigation of most crime is, however, conducted by the police acting principally under their common law powers and statutory powers of search and seizure and electronic surveillance, occasionally assisted in their investigation by the fruits of inquiries such

as those mentioned above. The police are entitled to question any person, whether suspected or not, in order to ascertain whether a crime has been committed, and if so, to discover the person who committed it. The police, while they are entitled to question suspects have, in general, no power however, to compel answers.

Notwithstanding the overlapping between s. 91(27) and s. 92(14), manifestly it would not be within provincial competence to enact legislation enabling a police officer to summon a suspect before an official and submit the suspect to compulsory examination under oath with respect to his involvement in a crime. Even though such legislation might be described as legislation in relation to the investigation of offences and thus appear to fall within the category of the administration of justice, such legislation in pith and substance would be legislation in relation to criminal procedure and thus within the *exclusive* competence of Parliament. In *A.-G. Que. and Keable*, *supra*, Estey J. said, at p. 257 S.C.R., p. 80 C.C.C., p. 192 D.L.R.:

> On the other hand, it is not only the Province and its agencies which may be concerned with the enforcement of the criminal law. It is equally clear that s. 92(14) does not authorize the Province to legislate with respect to criminal procedure directly or indirectly. It is the *Criminal Code* which sets forth the procedure prescribed by the sovereign authority, the Parliament of Canada, and which is to be followed in the investigation of crime and in the prosecution of ensuing charges. The Province, in the discharge of its role under s. 92(14) of *The British North America Act* may be required, or may find it convenient, to examine by the usual executive agencies or by a commission of enquiry, the operation of its policing facilities and personnel, and the prevalence of crime and its nature in the Province. Such was the case before the Court in *Di Iorio*, *supra*. At the other end of the scale, the enforcement agencies of the Province may of course investigate allegations or suspicions of

> specific crime with a view to the enforcement of the criminal law by prosecution. This investigation must be in accordance with federally prescribed criminal procedure and not otherwise, as for example, by coercive enquiry under general enquiry legislation of the Province.

He further said at p. 258 S.C.R., p. 81 C.C.C., p. 193 D.L.R.:

> One of the main bastions of the criminal law is the right of the accused to remain silent. In the coldest practical terms, that right, so long as it remains unaltered by Parliament, may not be reduced, truncated or thinned out by provincial action.

The views of Martin J.A. were expressly endorsed by the Chief Justice of Canada in delivering the majority judgment in Attorney General of Canada v. Canadian National Transporation Ltd. et al., 7 C.C.C. (3d) 449 at 475.

While the constitutional validity of the Order-in-Council is not in issue in this Court, it may be that it would have been vulnerable to question had the limitation not been imposed on the Commissioner that he not express any conclusions as to civil or criminal responsibility. This inquiry should not be permitted to become that which it could not have legally been constituted to be, an inquiry to determine who was civilly or criminally responsible for the death of the children or, in the circumstances of this case in lay language simply: who killed the children?

The Commissioner has found that the Order-in-Council allows him to determine whether or not the children died as a result of an overdose of digoxin or some other drug; to determine whether or not the administration of digoxin was accidental or non-accidental and to identify the administrator.

Relevant to the question in the stated case, at an early stage of the proceedings the Commissioner said of the appellants, Nelles and Trayner: "I cannot imagine that there could ever have been the slightest doubt as to why each of the members of the Trayner team is here represented by counsel funded for the Province. If such a doubt has ever existed, let me make it quite clear that each of them may be found to have been implicated, either by accident or with deliberation in the death of the children."

In his request for written argument from counsel, the Commissioner stated:

> There are two further matters which are not so urgent but I am satisfied must be resolved in the interest of a fair hearing. They are first the issue referred to above, namely whether I can in the Report if I should find that there was a deliberate overdose of Digoxin contributing to the deaths of any baby implicate any person in the overdose, or to put it in Mr. Scott's words, if I can "name names".

And, in a further statement:

> ...each of them (the members of the Trayner team) may be found to be implicated either by accident or by deliberation in the deaths of the children. (Emphasis added)

In his reasons for stating the case the Commissioner said:

> The problem simply stated is that if I could find upon the evidence that any child died from an unnatural cause, e.g. the administration of an overdose of digoxin (a finding I am clearly entitled to reach) and if the evidence also indicates that a certain person or persons administered the drug or participated in its administration either by accident *or design*, and I am entitled -- or indeed required -- to so state.

And he later observed:

> Our law does not give rise to either criminal or civil responsibility by the mere doing of an act. In criminal law, there must for a conviction be proved beyond a reasonable doubt (except in some few statutory provisions not applicable here) not only the act itself but a criminal intent in the mind of the actor. For the offence of murder or that of criminal negligence causing death, the two offences perhaps most likely to arise in the matter under consideration, the Criminal Code requires certain very specific and very special intents. Similarly in our civil law an act itself does not necessarily give rise to liability for damages or other relief to the person affected. It must be shown that the actor owed a duty and breached that duty and might reasonably have foreseen the damages that occurred. It follows that a finding by me of commission of the act either by accident or *by design* (even if the finding were that of a court of competent jurisdiction which it would not be) would not without more give rise to either criminal or civil responsibility.
>
> I do not intend to make any findings of fact respecting the state of mind of any person performing any act which might have criminal consequences nor do I intend to go into the duty owed by one person to another and whether or not that duty was breached. Nor do I intend to consider the defences that may be available under our law. I do, however, intend to study any evidence relating to the possibility of the administration of a Digoxin overdose (or indeed the evidence relating to any other act resulting in the unnatural death of any child) to determine where I can *whether or not the administration was accidental*, and where the evidence justifies it I intend to identify the administrator, and I shall answer the first question raised accordingly. (Emphasis added)

The majority of the Divisional Court answered the question in the affirmative. They reached this conclusion because the Commissioner said that he did not intend to make any findings of fact respecting the state of mind of any person performing an act which might have criminal consequences nor go into the duty owed by one person to another (the nurse to a patient) and whether the duty was breached. Further, he did not intend to consider the defences that might be available to any person. The majority concluded that the findings of fact which the Commissioner proposed to make would not amount to a conclusion forbidden by the Order-in-Council. They did, however, regard the distinction as a fine one but said that the case was one where fine lines had to be drawn.

In our opinion, in the Commissioner's terms of reference as stated by the Order-in-Council lies the answer to the question which he has stated. Clause 3 of the Order-in-Council directs the Commissioner to inquire into and report on and make any recommendations in respect to how and by what means the children came to their deaths. It is plain that the cause of death is the matter which the Commissioner is directed to inquire into and report upon and it is common ground that this means not only the question of whether death was caused by a fatal dose of digoxin but also whether the fatal dose was administered accidentally or otherwise. But the Order-in-Council specifically limits the

Commissioner by forbidding him to express any conclusion of law regarding civil or criminal responsibility for a death or the deaths. In our opinion, such a conclusion may be expressed by findings of fact which without more, when found against a named person, constitute a conclusion of criminal or civil responsibility. In the circumstances, if in carrying out the direction in the Order-in-Council the Commissioner, in determining the cause of death makes such findings, then he is by this specific limitation precluded from naming the persons whose act caused or contributed to the death or deaths of the children.

During the argument of the appeal emphasis was placed upon the care taken by the Commissioner to consider and weigh the several interests that are concerned with the death of the children in arriving at his conclusion as to what he will determine as set out in the question which he has stated. Those interests, of course, include the institution, the public, the parents and the persons who might be adversely affected by his decision. But with the greatest deference, the matter under consideration is not one in which the Commissioner has a discretion that can be the basis of determining what issue he will decide. The interests concerned were balanced when the Order-in-Council was made and the directions given to him as to what he was to inquire into and report upon subject to the specific limitations stated. He has no discretion that would permit him to decline to make a finding of intent or state of mind relative to the cause of death in order to make a finding identifying the person responsible because in his view of the interests it would be better to do so.

Further, the fact that the findings or conclusions made by the Commissioner are not binding or final in future proceedings is not determinative of what he will decide. What is important is that a finding or conclusion stated by the Commissioner would be considered by the public as a determination and might well be seriously prejudicial if a person named by the Commissioner as responsible for the deaths in the circumstances were to face such accusations in further proceedings. Of equal importance, if no charge is subsequently laid, a person found responsible by the Commissioner would have no recourse to clear his or her name.

It was contended that in making the Order-in-Council, regard was had to the provisions of the Coroners Act, R.S.O. 1980, c. 93, and in particular s. 34(1) and (2). It was submitted that the decisions of the courts with respect to proceedings under that and similar Acts should be considered and applied. In some cases referred to where the tribunal had reached a conclusion of law amounting to criminal or civil responsibility, the court said the tribunal ought not to have done so. In any event, we regard those cases as being of little assistance. This case is unique. There was an extensive police investigation and a prosecution that failed. The Attorney

General for the Province has clearly stated that if further evidence should be found there will be further prosecutions. He has stated and it is a fact "that there is no precedent for an inquiry of this nature into deaths thought to have been the result of deliberate criminal acts." In our opinion the specific limitation imposed on the Commission by the Order-in-Council in the circumstances was imposed out of concern for those persons who might become involved in other proceedings or be called upon to stand their trial. This concern for fairness is traditionally our way and so what we regard as a clear direction to the Commissioner in the Order-in-Council was struck accordingly and the cases referred to are of little help.

Turning to the question stated by the Commissioner. In it he has referred to actions which were accidental or otherwise than by accident. Elsewhere he has referred to non-accidental actions as actions by design or with deliberation or deliberate. In our opinion, a finding that death was caused by the deliberate actions of a named person being the administration of a toxic dose of digoxin, or that such actions were with deliberation or design really amounts to a finding that such person acted with intention to cause death and so a conclusion of law as to civil or criminal responsibility. Further, it is a reasonable inference that a person intends the natural consequences of his acts and such a finding as that referred to against a nurse in this case would leave nothing further to be said to amount to a conclusion forbidden by the Order-in-Council. This is particularly so if the same person were said to have so caused the deaths of more than one of the children.

In the result, then, we think the Divisional Court was wrong in its conclusion that the question stated by the Commissioner should be answered in the affirmative. To be clear, it is our opinion that if there is a finding of non-accidental administration of a lethal overdose of digoxin, thereby causing death, the Commissioner is prohibited from naming the person responsible for to do so would amount to stating a conclusion of civil or criminal responsibility. In addition, if the act of administration of a lethal dose of digoxin by a member of the staff of the hospital to a patient was "accidental", naming the person administering it would in the circumstances of this case also amount to a conclusion of civil or criminal responsibility and is prohibited. The Commissioner is obliged to hear all of the evidence relating to the cause of the death of the children and this would include evidence which tended to show that one or more of them died as a result of unlawful or negligent acts. While the Commissioner must not identify an individual as being legally responsible for a death, he should analyse and report upon all of the evidence with respect to the circumstances of each death and if he can, make recommendations with respect to that evidence.

It was probably inherent in the terms of the Order-in-Council that the task of meeting the "need of the parents and the public as a whole to be informed of all available evidence" by "full examination" of the matters to be inquired into and "to ensure full public knowledge of the completeness of the matters referred to", but to do so "without expressing any conclusion of law regarding civil or criminal responsibility", was one of extreme difficulty, at times approaching the impossible. Where such an impasse arises it should be resolved, in our opinion, by a course that best protects the civil rights of the persons the limitation was designed to protect.

The task of the Commission is thus a delicate and difficult one, but the limitation imposed by the Order-in-Council must be obeyed.

The appeal succeeds. The order of the Divisional Court is set aside and in its place an order will go that the question in the stated case be answered in the negative.

W. G C CJO G.A. Gale C.J.O.
John W JA John W. Brooke J.A.
J D J. Arnup J.A.
G A G.H. Martin J.A.

/ec L. W.

L.W. Houlden J.A.

IN THE SUPREME COURT OF ONTARIO

COURT OF APPEAL

BETWEEN:

SUSAN NELLES, PHYLLIS TRAYNER, THE REGISTERED NURSES ASSOCIATION OF ONTARIO and THIRTY-NINE REGISTERED NURSES AT THE HOSPITAL FOR SICK CHILDREN

Appellants
(Applicants)

-and-

THE HONOURABLE MR. JUSTICE S.G.M. GRANGE, COMMISSIONER

Respondent
(Respondent)

-and-

VARIOUS OTHER INTERESTED PARTIES

JUDGMENT

Released: April 12, 1984

C.J.O.

APPENDIX 4

No. 12976/82

IN THE SUPREME COURT OF ONTARIO

BETWEEN:

SUSAN NELLES

Plaintiff

- and -

HER MAJESTY THE QUEEN IN RIGHT OF ONTARIO, ATTORNEY GENERAL FOR ONTARIO, JOHN W. ACKROYD, JAMES CRAWFORD, JACK PRESS and ANTHONY WARR

Defendants

STATEMENT OF CLAIM

(Writ issued the 5th day of November, A.D. 1982).

1. The Plaintiff is a resident of the City of Belleville, in the Province of Ontario and a registered nurse qualified to practise in the Province of Ontario.

2. The Defendant John W. Ackroyd is Chief of Police of the Metropolitan Toronto Police Force and is responsible for the actions of the Defendants Crawford, Press and Warr pursuant to section 24(1) of the Police Act, R.S.O. 1980, c. 381, as amended.

3. The Defendant James Crawford is an Inspector in the Metropolitan Toronto Police Force.

4. The Defendant Jack Press is a Staff Sergeant in the Homicide Squad of the Metropolitan Toronto Police Force.

5. The Defendant Anthony Warr is a Sergeant in the Homicide Squad of the Metropolitan Toronto Police Force.

6. Robert McGee, Q.C. and Jerome Wiley, Esquire are barristers and solicitors licenced to practise in the Province of Ontario and at all material times were Crown Attorneys acting as agents of the Attorney General for Ontario.

7. On March 25, 1981, the Defendants Press and Warr, counselled, encouraged, aided and abetted by the Defendant Crawford, the Crown Attorneys McGee and Wiley, and the Attorney General for Ontario, arrested and charged the Plaintiff with four counts of first degree murder as follows: Janice Estrella on January 11, 1981; Kevin Pacsai on March 11, 1981; Allana Miller on March 21, 1981; and Justin Cook on March 22, 1981.

8. On March 25, 1981 and March 27, 1981, respectively, the Defendant Press, counselled, encouraged, aided and abetted by the Defendants Warr and Crawford, the Crown Attorneys McGee and Wiley, and the Attorney General for Ontario, appeared before Justices of the Peace and swore two informations that he believed and that he had reasonable and probable cause to believe that the Plaintiff did commit the said offences of first degree murder.

9. As a result of the said informations the Plaintiff was arrested on March 25, 1981, and wrongfully imprisoned and deprived of her liberty from the time of her arrest until March 30, 1981.

10. A preliminary inquiry into the said charges of first degree murder was commenced on January 4, 1982, in the Provincial Court (Criminal Division) for the Judicial District of York before His Honour Judge Vanek and on May 21, 1982, the Plaintiff was discharged on all counts.

11. The Defendants Press and Warr were negligent in laying the informations against, arresting, prosecuting and continuing to prosecute the Plaintiff on four counts of first degree murder and the Defendant Crawford, the Crown Attorneys McGee and Wiley, and the Attorney General for Ontario were negligent in counselling and encouraging the Defendants Press and Warr to lay the informations against and arrest the Plaintiff and in prosecuting and continuing to prosecute the Plaintiff. The following are the particulars of their negligence:

(a) they failed to conduct a proper and sufficient investigation;

(b) they laid the informations against the Plaintiff even though the investigation did not establish sufficient grounds for the laying of the charges;

(c) they acted on misinformation provided by the staff of the Hospital for Sick Children without verifying or substantiating the information;

(d) they continued to prosecute the Plaintiff after discovering that the Plaintiff did not have access to Janice Estrella and could not have been reasonably associated with the death of the Estrella child;

(e) they continued to prosecute the Plaintiff after learning from the autopsy of the Lombardo child that the Plaintiff could not have been associated with the death of that child;

(f) they laid the informations against the Plaintiff for the said counts of first degree murder in the absence of sufficient medical evidence upon which to base the charge; and

(g) they presented at the preliminary inquiry the Plaintiff's proper exercise of her lawful right to remain silent as evidence of her guilt.

12. The Plaintiff states that the Defendants violated sections 7, 11(c) and 11 (d) of the Canadian Charter of Rights and Freedoms by relying upon the Plaintiff's proper exercise of her lawful right to remain silent as evidence of her guilt in the laying of and prosecution of the said charges.

13. The Plaintiff relies upon section 24(1) of the Canadian Charter of Rights and Freedoms.

14. In the alternative, the Defendants Press and Warr, counselled, encouraged, aided and abetted by the Defendant Crawford, the Crown Attorneys McGee and Wiley, and the Attorney General for Ontario, did maliciously and without reasonable and probable cause swear two informations against and prosecute the Plaintiff with the said counts of first degree murder.

15. By reason of the laying of and the prosecution of the said charges, the Plaintiff was greatly injured in her credit, character and reputation, and has suffered mental and bodily pain and anguish, and has been put to considerable trouble, inconvenience, anxiety and expense, and has been greatly injured in her career and has thereby suffered damage.

16. By reason of the laying of and the prosecution of the said charges, the Plaintiff incurred legal costs for her defence in the amount of $104,850.15.

17. The Plaintiff therefore claims as against the Defendants and each of them:

(a) general damages for malicious prosecution, negligence, false imprisonment, and the infringement of the rights provided by sections 7, 11(c) and 11(d) of the

Canadian Charter of Rights and Freedoms in the amount of: $500,000.00;

(b) special damages in the amount of: $104,850.15;

(c) punitive, exemplary and aggravated damages in the amount of: $250,000.00;

(d) pre-judgment interest pursuant to section 36 of the Judicature Act, R.S.O. 1980, c. 223, as amended;

(e) solicitor and client costs; and

(f) such further and other relief as to this Honourable Court may seem just.

18. The Plaintiff proposes that this action be tried at the City of Toronto, in the Judicial District of York.

DELIVERED at Toronto, Ontario, this 26th day of January, 1983, by MESSRS. STIKEMAN, ELLIOTT, ROBARTS & BOWMAN, Barristers & Solicitors, P.O. Box 85, Suite 4900, Commerce Court West, Toronto, Ontario M5L 1B9, Solicitors for the Plaintiff.

rec'd 26/1/83
S. Lockett [illegible] at 3:30

No. 12976/82.

IN THE SUPREME COURT OF ONTARI

(ACTION COMMENCED IN THE JUDICIAL DISTRICT OF YORK)

BETWEEN:

SUSAN NELLES

Plaintiff,

- and -

HER MAJESTY THE QUEEN IN RIGHT OF ONTARIO, ATTORNEY GENERAL FOR ONTARIO, JOHN W. ACKROYD, JAMES CRAWFORD, JACK PRESS and ANTHONY WARR

Defendants.

STATEMENT OF CLAIM

MESSRS. STIKEMAN, ELLIOTT,
ROBARTS & BOWMAN
Barristers & Solicitors
P.O. Box 85, Suite 4900
Commerce Court West
Toronto, Ontario
M5L 1B9

Solicitors for the Plaintiff.

APPENDICES 5, 6, 7, AND 8

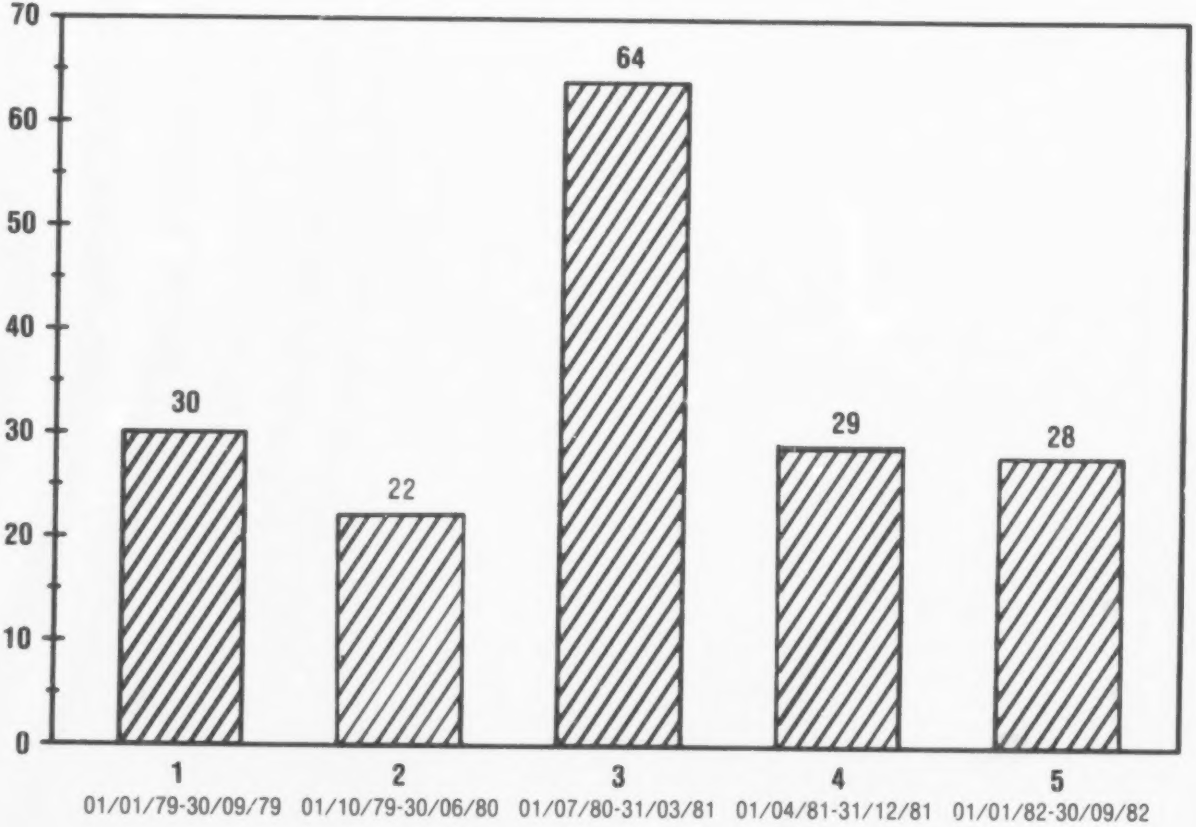
In all these graphs, period one refers to January 1 1979 to September 30 1979, period two refers to October 1 1979 to June 30 1980, period three refers to July 1 1980 to March 31 1981, period four refers to April 1 1981 to December 31 1981, period five refers to January 1 1982 to September 30 1982.

The time span referred to in Appendix 7 divides the twenty-four hour day into four six-hour segments. The time span referred to in Appendix 8 divides the twenty-four hour day into six four-hour segments. Within each time span, all five periods as explained above appear in different colours.

Appendix 5

TOTAL DEATH BY PERIOD

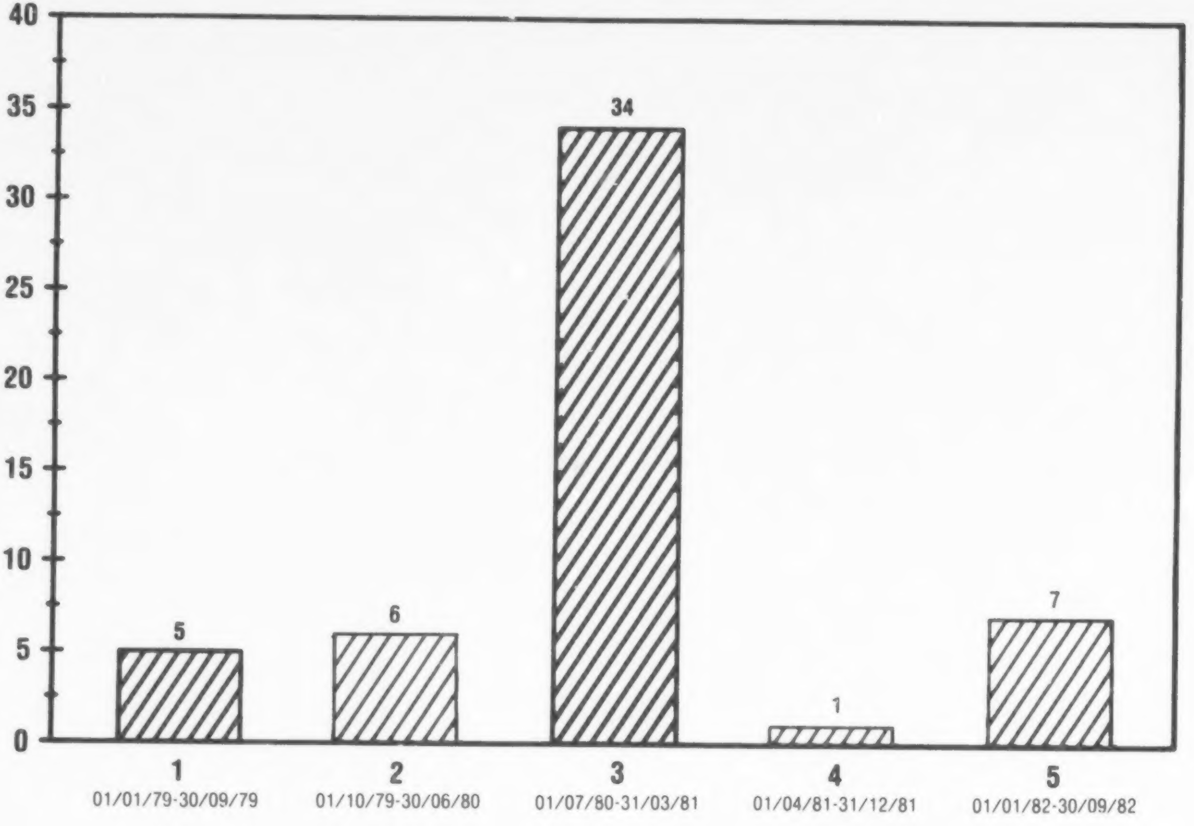
EACH PERIOD = 9 MONTHS



Appendix 6

ON-WARD DEATHS BY PERIOD

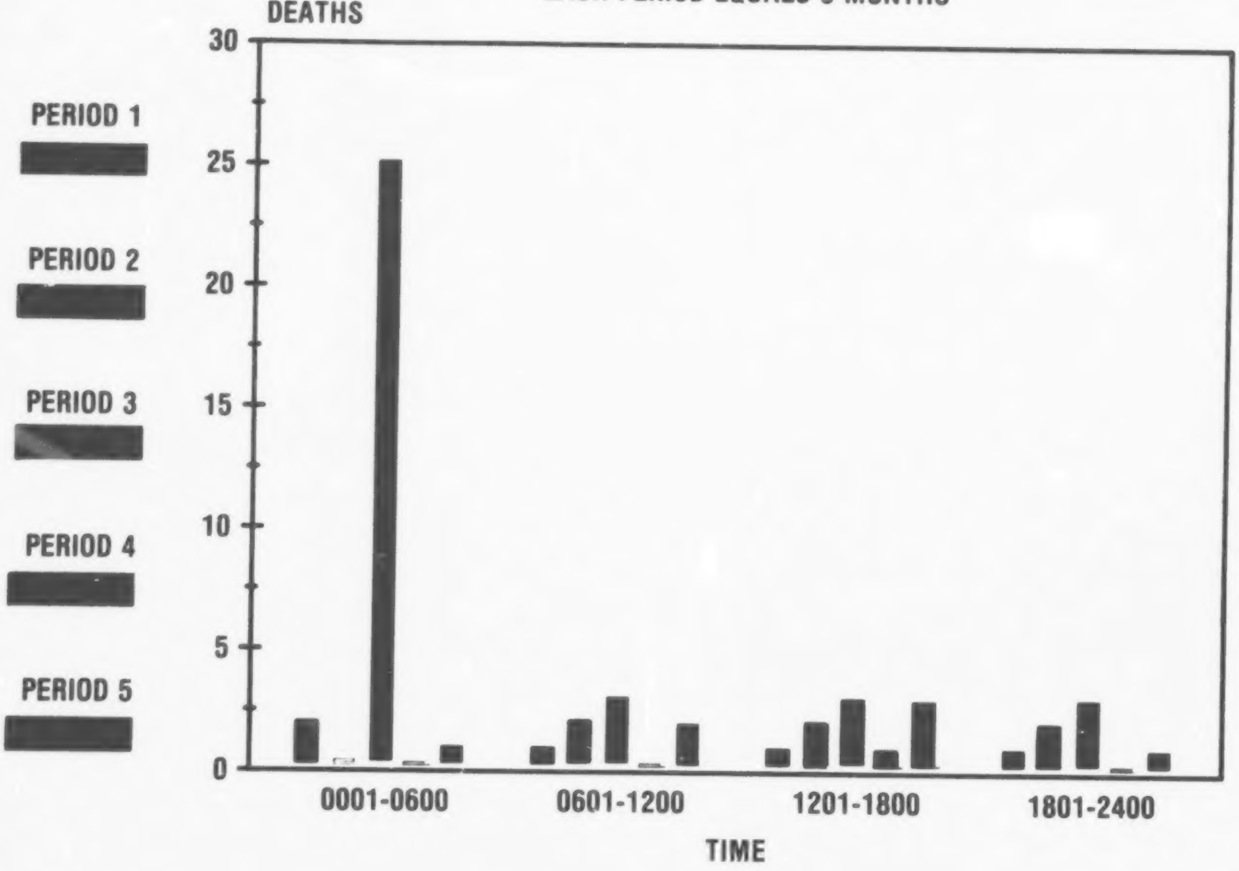
EACH PERIOD = 9 MONTHS



Appendix 7

ON-WARD DEATHS BY TIME

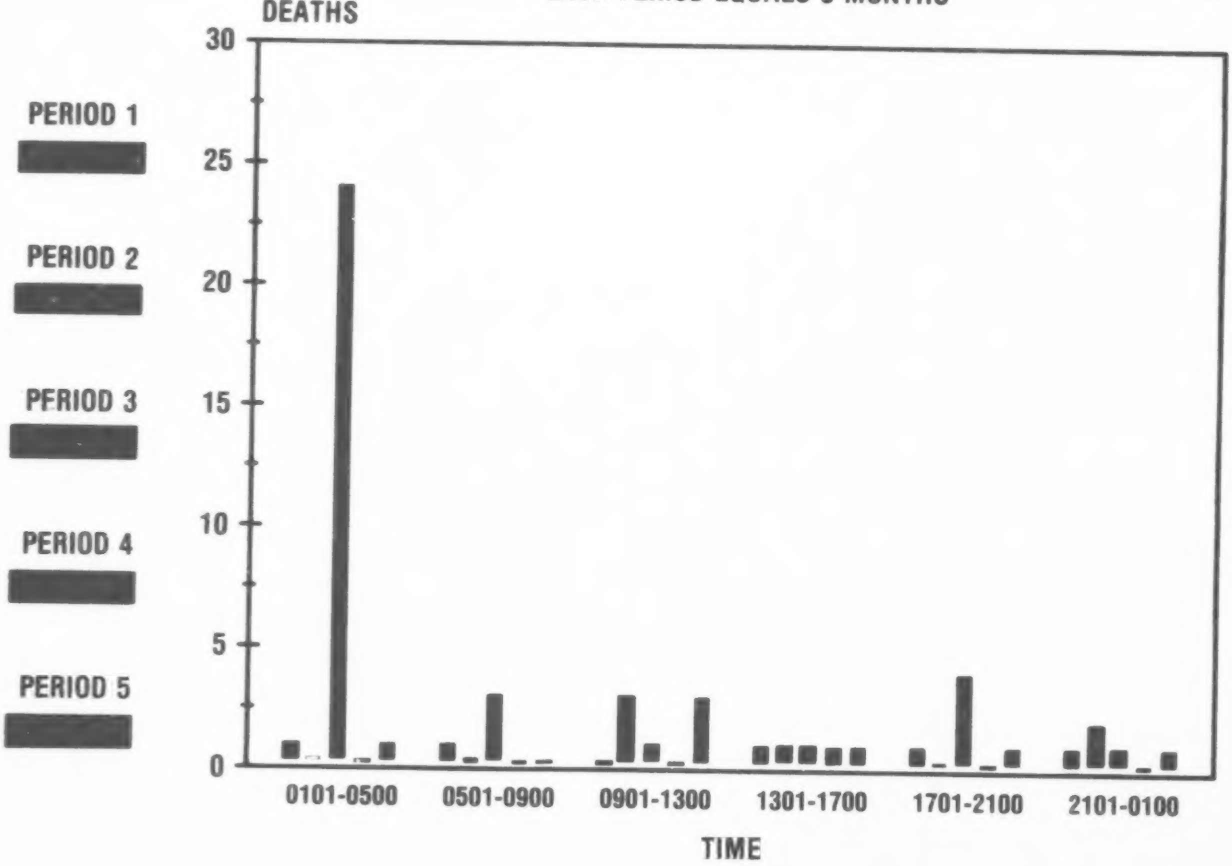
EACH PERIOD EQUALS 9 MONTHS



Appendix 8

ON-WARD DEATHS BY TIME

EACH PERIOD EQUALS 9 MONTHS



APPENDIX 9

SUMMARY OF ATLANTA REPORT FINDINGS

NAME	DEATH					DIGOXIN			
	EXPECTED	UNEXPECTED	CONSISTENT WITH CLIN. CONDITION	INCONSISTENT WITH CLIN. CONDITION	CATEGORY	CONSISTENT WITH TOXICITY	INCONSISTENT WITH TOXICITY	SPECIAL CONCERN RE TOXICITY	SCORE
WOODCOCK		X		X	A	X		X	1
PERREAULT	X		X		C		X		1
BILODEAU	X		X		B	X			1
TAYLOR		X	X		A	X		X	1
DAWSON		X		X	A	X			1
HOOS		X	X		B	X			1
TURNER		X	X		A	X		X	1
SHRUM		X	X		C	X			1
MONTEITH	X		X		B	X			1
MURPHY	X		X		C		X		1
VELASQUEZ		X		X	A	X		X	1
HEYWORTH	X		X		C		X		1
GAGE		X	X		B	X			2
MCKEIL		X	X		B	X			1
ADAMO		X	X		A	X		X	1
VOLK		X	X		C		X		1
LUTES		X	X		B	X			1
ONOFRE		X		X	A	X		X	1
MACDONALD		X	X		A	X		X	1
GOSSELIN		X		X	A	X			1
LOMBARDO		X	X		A	X			4
BELANGER		X		X	A	X			3
ESTRELLA		X	X		A	X		X	2
FAZIO	X		X		B	X			1
FLORYN	X		X		C		X		1
THOMAS		X	X		B	X			1
LEITH	X		X		C	X			1
WARNER		X	X		B	X			1
HINES	X		X		A		X		3
GIONAS		X	X		A	X		X	2
MANOJLOVICH		X	X		A	X		X	1
PACSAI		X		X	A	X		X	4
INWOOD		X	X		A	X			4
GARDNER	X		X		B	X			1
MILLER		X	X		A	X			4
COOK		X	X		A	X		X	5

APPENDIX 10

APPENDIX TO CHAPTER 7 ON EXPERT WITNESSES

NAME	POSITION AS AT MARCH, 1981 OR AT RELEVANT TIME
ATLANTA REPORT AUTHORS (See: BUEHLER, Dr. James KUSIAK, Robert SMITH, Dr. Lesbia WALLACE, Dr. Evelyn)	
BAIN, Dr. Harry	Former Chief of Pediatrics Hospital for Sick Children
BECKER, Dr. Laurence	Senior Pathologist Hospital for Sick Children
BUEHLER, Dr. James	Medical Epidemiologist Field Services Division Centers for Disease Control Atlanta, Georgia
CARVER, Dr. David	Chief of Pediatrics Hospital for Sick Children
CIMBURA, George	Director of the Toxicology Section Centre of Forensic Sciences, Toronto
COSTIGAN, Dr. Colm	Chief Pediatric Resident Hospital for Sick Children
CUTZ, Dr. Ernest	Senior Pathologist Hospital for Sick Children
deSA, Dr. Derek*	Chief of Pathology Winnipeg Children's Hospital Winnipeg, Manitoba

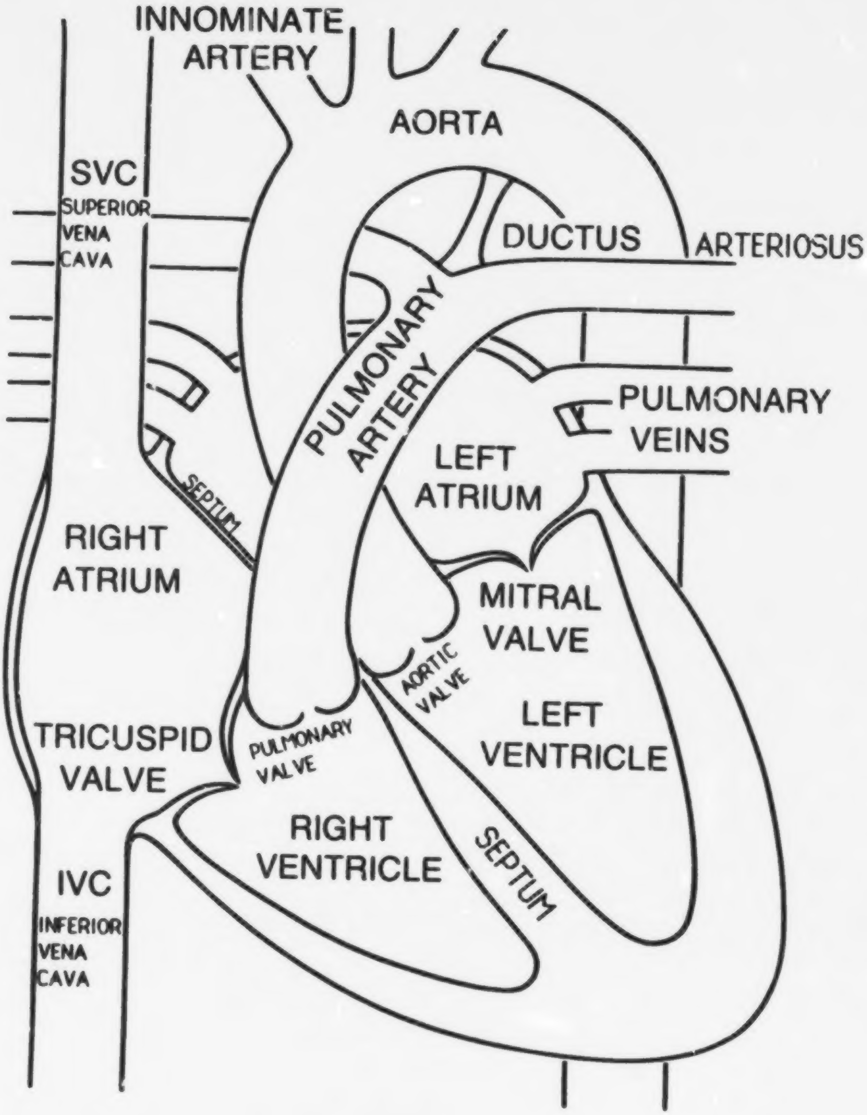
* Did not testify

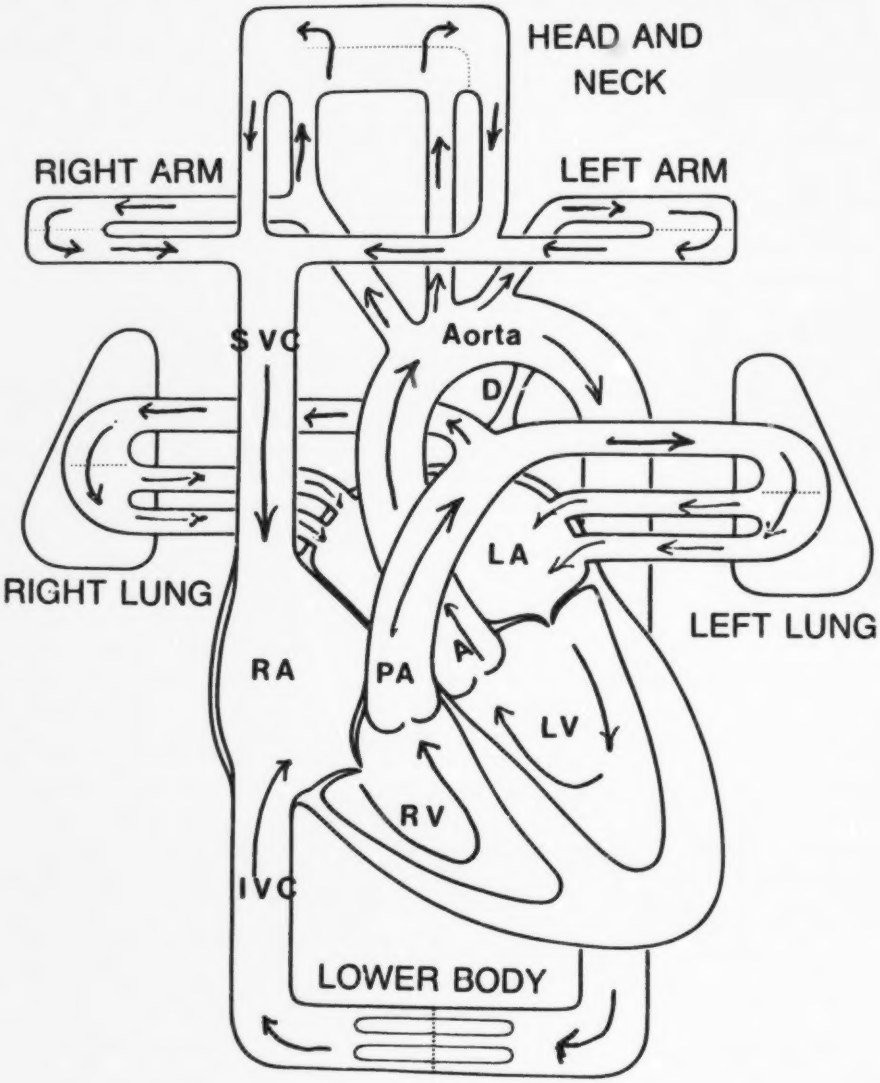
NAME	POSITION
ELLIS, Dr. Graham	Assistant Biochemist Hospital for Sick Children
FAY, Dr. John	Cardiologist Department of Medicine and Department of Pediatrics Kingston General Hospital
FOWLER, Dr. Rodney	Senior Cardiologist Hospital for Sick Children
FREEDOM, Dr. Robert	Senior Cardiologist Pathologist Hospital for Sick Children
HASTREITER, Dr. Alois	Director of the Division of Pediatric Cardiology University of Illinois Hospital Chicago, Illinois
IZUKAWA, Dr. Teruo	Cardiologist Hospital for Sick Children
KANTAK, Dr. Anand	Pediatric Resident Hospital for Sick Children
KAUFFMAN, Dr. Ralph	Director of Clinical Pharmacology and Toxicology Children's Hospital Detroit, Michigan
KOBAYASHI, Dr. Jeffrey	Pediatric Resident Hospital for Sick Children
KUSIAK, Robert	Biostatistician Government of Ontario
MACKLEM, Dr. Peter	Physician-in-Chief Royal Victoria Hospital McGill University Dean of Medicine Medical School Montréal, Québec

NAME	POSITION
MacLEOD, Dr. Stuart	Director of the Division of Clinical Pharmacology Hospital for Sick Children
MANCER, Dr. Kent	Senior Pathologist Hospital for Sick Children
McGEE, Dr. Marian	Associate Dean Faculty of Health Sciences Schools of Medicine, Nursing and Human Kinetics University of Ottawa
MIRKIN, Dr. Bernard	Director of the Division of Clinical Pharmacology Professor of Pediatrics and Pharmacology University of Minnesota Medical School Minneapolis, Minnesota
NADAS, Dr. Alexander*	Chief Emeritus Cardiology Children's Hospital Medical Center Boston, Massachusetts
PHILLIPS, Dr. James	Pathologist-in-Chief Hospital for Sick Children
ROSE, Dr. Vera	Cardiologist Hospital for Sick Children
ROWE, Dr. Richard	Director of the Division of Cardiology Hospital for Sick Children
SECCOMBE, Dr. David	Assistant Medical Biochemist Shaughnessy and Vancouver General Hospitals

* Did not testify.

NAME	POSITION
SMITH, Dr. Lesbia	Senior Medical Consultant Environmental Health Ontario Ministry of Health
SOLDIN, Dr. Steven	Associate Biochemist Hospital for Sick Children
SPIELBERG, Dr. Stephen	Pharmacologist Hospital for Sick Children (since July/81)
WALLACE, Dr. Evelyn	Field Epidemiologist Ontario Ministry of Health


INNOMINATE ARTERY
AORTA
SVC
SUPERIOR VENA CAVA
DUCTUS ARTERIOSUS
PULMONARY ARTERY
PULMONARY VEINS
LEFT ATRIUM
SEPTUM
RIGHT ATRIUM
MITRAL VALVE
AORTIC VALVE
LEFT VENTRICLE
TRICUSPID VALVE
PULMONARY VALVE
RIGHT VENTRICLE
SEPTUM
IVC
INFERIOR VENA CAVA


HEAD AND NECK
RIGHT ARM
LEFT ARM
SVC
Aorta
D
RIGHT LUNG
LA
LEFT LUNG
RA
PA
A
LV
RV
IVC
LOWER BODY

APPENDIX 13

These definitions do not pretend to be exact: they are designed solely to help the non-medical reader.

GLOSSARY

MEDICAL TERM	DEFINITION
AV block	Atrioventricular heart block, i.e., dissociation of heart beats between the atrium and the ventricle.
alpha half life	Time in which half the digoxin in the blood is distributed to tissue.
alpha phase	The period of distribution of digoxin to blood and tissue.
aorta	The main artery taking blood from the heart to other parts of the body.
aortic arch	The curve between the ascending and descending aorta.
apex	Used in the Hospital as a synonym for heart rate.
apnea	Cessation of breathing for a short period.
apnea monitor	Device which sounds an alarm if breathing ceases for more than the preset period of time.
arhinencephaly	Absence of a portion of the brain.
arrhythmia	Irregular heart beat.
arterioles	Very small blood vessels.
artery	A blood vessel carrying blood away from the heart; the "great arteries" are the aorta and the pulmonary artery.
ascitic fluid	Fluid which accumulates in the peritoneal cavity.
asystole	Absence of contraction of the heart.
atresia	Closing.
atretic	Closed or almost closed.

GLOSSARY

MEDICAL TERM	DEFINITION
atrial flutter	Rapid rhythmic atrial contractions.
atrial septal defect	A hole in the septum dividing the atria.
atrioventricular defect	Defect affecting both atrium and ventricle.
atrioventricular node	A part of the heart which transmits electrical impulses from atrium to ventricle.
atrium (pl. artria)	One of the upper chambers of the heart.
atrophy	Waste or grow smaller in size.
beta half life	Time in which half the digoxin is eliminated from the body.
beta phase	Elimination phase of digoxin from the body.
Blalock-Taussig shunt	An operation which links the artery going to the right arm with the right pulmonary artery.
blue baby condition	Lack of oxygen in the blood causing cyanosis (blueness) in the infant.
blue spell	Period of extreme cyanosis caused by contractions of the heart muscle when the blood is underoxygenated.
bolus dose	Drug administered rapidly through the intravenous line.
bronchiolitis	Bronchopneumonia.
bradycardia	Slow heart rate.
brown fat	A form of fat seen before birth but normally disappearing soon after.
buretrol	Medication chamber which forms part of the intravenous apparatus.
cardiac arrest	Cessation of activity of the heart.

GLOSSARY

MEDICAL TERM	DEFINITION
cardiac catheterization	An investigative and diagnostic procedure whereby a catheter is passed through the veins into the heart.
cardiac monitor	Device which sounds an alarm if the heart rate goes above or below preset limits.
cardiomegaly	Enlarged heart.
cardiomyopathy	Chronic muscle disease of the heart.
cardiopulmonary arrest	Both cardiac and respiratory arrest.
cardiopulmonary resuscitation	Compression of the chest to stimulate the heart to resume beating.
coarctation	Narrowing.
coarctectomy	Elimination of coarctation.
conduction system	Elements of the heart which transmit the electrical impulses.
crash cart	Cart containing drugs and equipment needed in resuscitation.
cyanosis	Blue colour indicating a lack of oxygen in the blood.
dextrocardia	Heart displaced to the right.
dextroposition	Displaced to the right.
diaphoretic	Perspiring heavily.
diaphragm	The partition of muscular membrane separating the abdominal and chest cavities.
DiGeorge syndrome	Deficiency of the immune system.
digitalizing dose	First three large "loading" doses of digoxin.
diuretic	Medication to induce urination.
Down's syndrome	Mongolism, chromosomal disorder causing mental retardation.

GLOSSARY

MEDICAL TERM	DEFINITION
drug screen	Series of laboratory tests to determine the existence of certain drugs in blood or tissue.
ductus	A channel or passage.
ductus arteriosus	A channel or passage between the great arteries of the heart.
dysmorphic	Malformed or unusual.
dysrhythmia	Defective rhythm.
echocardiogram	An examination of the heart performed by sound waves.
ectopic	Misplaced.
edema	Swelling.
edema fluid	Fluid forming within the tissue cells causing swelling.
electrocardiagram	Tracing of the electrical activity of the heart.
endocardial fibroelastosis	A thickening of the tissue wall of the ventricle.
enterocolitis	Inflammation of the intestines.
Escherichia coli (E.coli)	Micro-organisms in the blood.
extra medullary hematopoiesis	Formation of red blood cells outside the bone marrow.
fixed tissue	Tissue which has been placed in a preservative solution.
foramen ovale	An opening between the atria existing before birth which usually closes after birth.
FPIA	Fluorescent Polarization Immunoassay: a test which can be used to determine the digoxin level.
gastroenteritis	Inflammation or infection of the intestinal tract.
gastromalacia	Softening of the stomach wall.
GC/MS	Gas chromatography and mass spectrometry: a test which can be used to determine digoxin levels.

GLOSSARY

MEDICAL TERM	DEFINITION
gliosis	Scars in the brain stem.
gross autopsy	Autopsy minus microscopic or laboratory studies.
gutter blood	Blood from the pelvic cavity.
heart block	See AV block.
heart murmur	Unnatural sound heard in the heart.
hemodynamic	Pertaining to circulation of the blood.
hemolyse	Separation of hemoglobin from the red blood cells.
hemophilia	A condition which produces excessive bleeding.
HPLC	High pressure liquid chromatography: a test which is designed to separate digoxin from digoxin-like substances.
hydrocephalus	Accumulation of fluid in the skull.
hyperkalemia	Excess of potassium in the blood.
hypertension	High blood pressure.
hypertrophy	Overdevelopment.
hypokalemia	Insufficiency of potassium in the blood.
hyponatremia	Deficiency of sodium in the blood.
hypoplastic left heart OR right heart	Incomplete development of that side of the heart.
hypothermia	Low temperature.
hypoxia	Shortness of oxygen.
ichthyosis	A congenital skin disease causing hardening.
infarct	Death of an area of tissue.
inferior vena cava	Vein carrying blood to the heart from the lower body.
ischemic encephalopathy	Brain damage arising from a deficiency of blood.
jaundice	A yellowish condition of the skin often associated with liver disease.

GLOSSARY

MEDICAL TERM	DEFINITION
junctional rhythm	A regular heart rhythm originating in the atrioventricular node rather than in the sinus node.
ligation	The tieing of or closing off.
loading dose	One of three large or "digitalizing" doses of digoxin.
maintenance dose	Normal post-digitalization dose of digoxin.
mEq	Milliequivalent. The number of grams of a substance in one millilitre of a solution.
missed-SIDS	A SIDS episode not resulting in death.
mitral valve	The left atrioventricular valve.
mongolism	Chromosomal disorder causing mental retardation (Down's syndrome).
myelomeningocele	Protrusion of the spinal cord through a defect in the spinal column.
myocardial	Relating to heart muscle.
nanogram	A billionth of a gram, a form of digoxin level measurement.
nanomole	A billionth of a mole, another form of digoxin level measurement.
naso-gastric tube	Tube inserted through the nostril into the stomach for feeding.
necrosis (tissue)	Death of tissue.
ng/g	Nanograms per gram.
ng/ml	Nanograms per millilitre.
occlude	Close or shut.
pacemaker	A device to stimulate electrical impulses within the heart. The sinus node is the natural pacemaker.

GLOSSARY

MEDICAL TERM	DEFINITION
palliative surgery	Surgery which relieves but does not cure.
paroxysmal atrial tachycardia	Spasms of very rapid heart beat.
patent	Open.
plasma	Serum including the clotting element of the blood.
pneumonectomy	Removal of a lung.
pneumonitis	Acute inflammation of the lungs.
polyspleenia	Multiple spleens.
pulmonary atresia	A situation in which the pulmonary valve is closed.
pulmonary collapse	Collapse of the lungs.
pulmonary stenosis	Narrowing of the pulmonary valve.
renal	Relating to the kidney.
respiratory arrest	Cessation of breathing.
RIA	Radioimmunoassay: a test which can be used to determine digoxin levels.
sage pump	Device to regulate the flow rate of intravenous fluid and medications.
sagittal sinus	A collection of veins in the brain.
sepsis	Infection.
septum	A dividing wall or partition.
serum	The clear portion of the blood which separates from the clotting portion.
shunt	A passage or connection between two channels in the body.
shunt occlusion	Closing of shunt.
sick sinus syndrome	Defect in the conduction system of the heart.
SIDS	Sudden infant death syndrome, unexplained infant death, crib death.
sinus node	Transmitter of electrical activity within the heart.

GLOSSARY

MEDICAL TERM	DEFINITION
sinus rhythm	Normal heart rhythm.
stenosis	Narrowing (particularly of a valve).
subclavian artery	Artery which lies under the collar bone.
subendocardial infarct	Death of a portion of heart tissue.
superior vena cava	The vein carrying blood from the head, neck and arms.
systole	State in which the heart is contracted.
tachycardia	Rapid heart rate.
tachypnea	Rapid breathing.
tetralogy of Fallot	A combination of four cardiac defects including pulmonary stenosis, one or more ventricular septal defects, dextroposition of the aorta, and right ventricular hypertrophy.
transient adrenal insufficiency	A brief episode of adrenal gland failure.
transposition of the great arteries	Reversal of the aorta and pulmonary arteries.
tricuspid valve	Right atrioventricular valve.
truncus arteriosus	A condition in which only one large artery leaves the heart.
valvotomy	Incision or opening of a valve.
vein	A blood vessel carrying blood to the heart.
vena cava	A vein which empties into the heart.
ventilator	Device to assist breathing.
ventricle	One of the lower chambers of the heart.
ventricular atrial shunt	A passage between the ventricles of the brain and the right atrium of the heart.
ventricular fibrillation	Rapid uncontrolled contractions of the ventricles.

GLOSSARY

MEDICAL TERM	DEFINITION
ventricular flutter	A form of rapid ventricular tachycardia.
ventricular septal defect	A hole in the wall of the heart separating the two ventricles.
vital signs	Heart rate, blood pressure, rate of respiration, and temperature.
Waterston shunt	An artificial channel from the aorta to the pulmonary artery.

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